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A CASE OF MULTIPLE FIBROMATA OF THE NERVES, WITH ARTHRITIS DEFORMANS.[1]

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and
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of Chicago.

Clinical History (Dr. Preble). Patient, female, J. S., aged thirty-five years. Comes from a family whose history presents nothing of interest, especially is there no history of any tumors in any way resembling the tumors presented by the patient. When very young, a tumor appeared on the forehead and grew slowly until the resulting deformity led to efforts of removal by some sort of a morcellating operation. Shortly after this other tumors appeared irregularly and in great numbers over the entire body.

The personal history gives nothing else of interest until young womanhood is reached, when the patient begins to suffer from pain and weakness, which gradually increased until they caused complete disability. She gives a history of a complete paraplegia without bladder or rectum disturbances. From the history given I am inclined to believe that the paraplegia was hysterical.

Some years after the appearance of the tumors, but just when the patient cannot recall, she began to show the beginning of the joint changes which are now so marked. These consist of swelling and deformity, but there is not much pain. The joints first and most involved were the small joints of the hand; the changes began symmetrically.

The tumors have not caused much disturbance as a rule, only a few of them having been painful. The patient states that they have varied in size from time to time, and that some of them have entirely disappeared spontaneously. Two years before I saw her a tumor on the left thigh ulcerated, and in spite of every treatment has never healed.

[1] Read before the Association of American Physicians, May, 1900.

When first seen, in 1894, the patient complained of abdominal pain, described as drawing in character and tending constantly to pull her forward. She complained also of pain in her legs and feet. It was difficult for her to get from her bed to her chair, and walking was impossible. The ulcer on the thigh was painful.

EXAMINATION. Small, emaciated and deformed woman; mind is clear; intelligence fair, although she is irritable and unreasonable. The skin is pale without any areas or flecks of pigmentation. It is marked by many tumors, which vary in size and appearance. On the forehead are small tumors up to the size of a small bean, arranged in irregular and tortuous strings, so that they give through the skin a sensation not unlike that of a scrotal varicocele. They are not painful or tender. They extend not only across the forehead, but back, also, into the hair. There are other tumors on the face, but none presenting this racemose arrangement. The tumors of the skin over the neck, body, and limbs vary in size from a large pinhead to the size of a nut. Some are sessile, some are pedunculated. They are covered usually by a thin and wrinkled skin, although some of them are quite smooth and shiny. None are pigmented or hairy; none are painful.

In the deeper parts we find fusiform or ovoid tumors arranged parallel to the long axis of the neck or limb. They permit of lateral but not longitudinal motion. They are not attached to the skin, and are not painful, with the exception of one in the neck.

On the posterior surface of one thigh is an ulcer half an inch in diameter and more than that in depth, with sharply cut borders. It exactly resembles the perforating ulcers of tabes.

The hands are greatly deformed; deformity is symmetrical, which began in the small joints. The joint surfaces are destroyed and the capsules greatly relaxed. The resulting deformity is as great but not exactly like the deformity seen in well-marked arthritis deformans. The wrist-joints are similarly affected, the knee and hip-joints are ankylosed, the motion of the jaw is limited, and there is considerable deformity of the spine.

The organs of the thorax and abdomen are normal; the blood shows merely a moderate degree of anæmia; pelvic examination under anæsthesia shows a virgin uterus, and high up in the pelvis there are tumors resembling those on the limbs.

The nervous system presents no alterations whatever. All the cranial nerves are in perfect condition; all the elements of the sense of touch are perfect. There are no paralyses; the leg reflexes cannot be examined because of the ankylosed joints.

As time went on the patient became gradually worse, and the deformity of the spine rapidly increased until the chin almost came in contact with the pubes. She continued to amuse herself as she had for years by making little trinkets and fancy articles, doing this in spite of the deformity of the hand. The pain in the abdomen and the legs continued; no paralyses or disturbances of the bladder or rectum appeared. About ten days before death gangrene of the toes of the left foot appeared. Death occurred without any apparent definite cause.

POST-MORTEM EXAMINATION (Dr. Hektoen). Female body, five feet three inches, slight rigor, extreme and uniform emaciation; the body is greatly deformed on account of a marked kyphoskoliosis, whose greatest curve lies in the lumbosacral spine, with its convexity to the right

and backward; in the cervical region is a smaller curve with the convexity to the left. The knee-joints are fixed at right angles, and the head of the tibia partly dislocated backward. Both hip-joints are immobile, and the thighs are flexed on the abdomen. The shoulder and elbow-joints are stiff. The left wrist-joint and the right ulnocarpal joint are apparently completely disorganized and flail-like; also the joints of the thumbs, the distal phalanges of which are flexed outward at right angles. There is palmar dislocation of all the phalanges of both hands, more marked in the left. The phalangeal joints of the first and fourth fingers of the right hand are flail-like—second and third fingers are not changed. The small joints of the left hand are changed to less degree. The nails of the fingers are not changed. The ankle-joints and the joints of the feet are changed in a similar manner. The skin of the toes of the right foot is dry, covered by scales, toe ends glossy, the nails greatly thickened and furrowed. The toes and dorsum of the left foot are the seat of a moist, foul gangrene. Upon the inner surface of the right leg is an oval area of furrowed, horny thickening of the skin, raised 5 mm. above the general surface, 6 x 3 cm. in extent. Over the tuberosity of the right ischium is a deep, oval sore.

The skin shows also innumerable tumors, varying in size from a pinhead to a pecan-nut, quite movable and unattached to deeper structures, some barely raised above the surface, some almost pedunculated. Over some the skin is smoothly drawn, over others it is laxed and wrinkled; they are either round and globular or oval and flattened; in many places they show a disposition to arrange themselves in lines; in the forehead and scalp they have a plexiform arrangement; they are probably most numerous in the skin of the face and neck, the largest one being situated in the subcutaneous tissue of the right side of the neck; they are also very numerous in the skin of the hands, especially the right; over the trunk they occur quite richly, occurring in parallel lines along some of the ribs; the skin of the legs contains the smallest number.

The peritoneal cavity is empty, its lining smooth and shining. The stomach is more vertical than usual, the lowest limit of the large curvature reaching below the umbilicus; the cæcum and vermiform appendix lie in the true pelvis, pressing the uterus and ovaries against the lateral and anterior walls and the bladder; the transverse colon describes a large curve, the convexity reaching to the lowermost part of the cavity. The diaphragm reaches to the fifth rib on both sides.

Both pleural cavities are empty, the pleuræ smooth. The right pleural cavity becomes very small in the lower part. Running along the lower edge of some of the ribs are extra-pleural rows of small nodules.

The phrenic nerve presents a few small cylindrical enlargements as it runs over the pericardium, which is empty and smooth.

There is no fat in the subserous tissue anywhere.

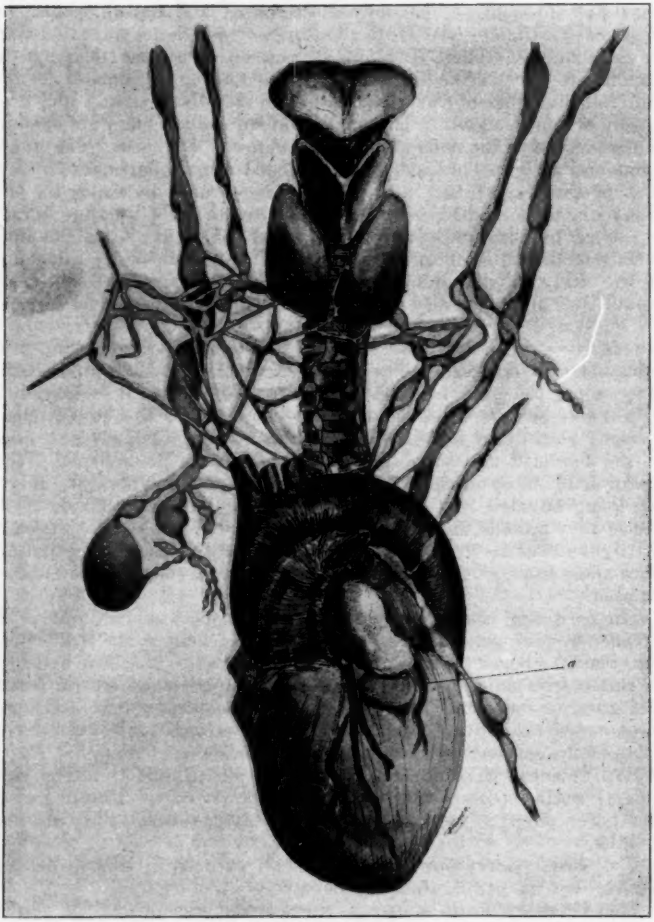
The tongue, pharynx and œsophagus are quite normal. The larynx and trachea are also normal. The thyroid is normal in size, the thymus absent.

The lungs crepitate; they are rather light and spongy; the cut surface is not much pigmented and is rather bloodless; there is no bronchitis; the peri-bronchial lymph-glands are not enlarged.

The pericardium is smooth; in the subpericardial tissue of the ante-

rior surface of the left venricle, just below the auriculo-ventricular sulcus, lies a rounded mass, 1.5 x 7 cm., over which the epicardium is a

FIG. 1.



Showing tumors of pneumogastric and cervical sympathetic nerves and their branches. At *a* subpericardial tumor nodule.

little roughened (Fig. 1). The heart is small, the coronary vessels convoluted, the myocardium brownish, the endocardium smooth.

The spleen is of the usual size and free from changes.

The liver is small, has nutmeg cut surface. The gall-bladder is empty. In the mesentery appear many small, whitish, flattened, and oval, firm bodies, many of which are in the direct course of small nerves. In the part near the root are softer, yellowish masses as large as split pease or so. The mesenteric attachment of the duodenum contains several pairs of small bodies closely together, one of which is whitish and rather firm, the other more yellowish and rather soft. The serous covering of the duodenum has numerous slightly raised patches of irregular outlines and of a peculiar saffron color; it is in general somewhat thicker and rougher than normal.

The stomach contains a quantity of blackish fluid; in the fundus an extensive district of the mucosa has been digested, and a small part of the wall is so soft that it ruptures on handling; otherwise the gastric mucosa is quite normal. The mucous membrane of the duodenum and jejunum contains a few irregular districts which are finely nodular to the feel, the membrane being somewhat granular to the eye. The ileum and the large intestine are quite normal, the latter containing masses of soft, yellow feces, the former being empty.

The nerves of the retroperitoneal tissue, including those radiating from the splanchnic ganglia, present closely aggregated beaded enlargements varying in size from a pinhead to a bean. The nerves in the pelvis behind the peritoneum present numerous globular and oval enlargements up to the size of walnuts, of a similar glistening and whitish appearance. Situated upon the inner surface of the left side of the upper half of the sacrum is a firm spherical mass as large as a pigeon's egg, which is deep bluish-black in color with a few gray spots, and which has a somewhat reticulated cut surface. The deep and superficial inguinal glands, especially on the right side, are pseudomelanotic and somewhat enlarged as well as quite firm.

The adrenals are normal in appearance, the right being rather flattened; there are no tumors in their immediate vicinity. The kidneys are small, the capsules free, the surface slightly granular in places and rather yellowish. The cortical markings are somewhat indistinct. The pelves, ureters, and urinary bladder normal. The ovaries and uterus are small; otherwise no changes.

The subcutaneous tissue of the scalp contains tangled, cord-like masses of thickened nerves both in the frontal and occipital regions; in many the thickening seems diffused, in other cases small white tumors spring from the epineurium.

The skull is of moderate thickness but increased density. The skull form is symmetrical. The dura is smooth. The pia contains a moderate amount of clear fluid. Externally the brain and the intracranial portion of the cerebral nerves are normal. The brain is hardened unopened in formalin solution. The right optic nerve and the right globe and motor nerves show no changes. The hypophysis is of about normal size and the cella turcica is rather deep. The sphenoidal sinuses are empty and smooth. The frontal sinuses are covered with a layer of dirty, grayish, thick fluid.

On account of the spinal curvature it is rather troublesome to remove the spinal cord, which seems of natural size; its membranes are smooth. Upon three or four of the lower dorsal nerves are small fusiform enlargements of the intraspinal portions. Upon each of the third and fourth right cervical nerves is a smooth, glistening, cylindrical enlarge-

ment, located within the spinal canal underneath the pia (Fig. 8); there are numerous similar enlargements in the course of the nerves outside of the canal; the larger of the intraspinal tumors is 3 by 1.5 cm., smooth and white, and presses the cord to one side; coursing over its surface are small, apparently healthy nerve bundles.

The spinal cord and brain are hardened as a whole in 4 per cent. formalin (see below).

In addition to the tumors in connection with the nerves already referred to, it is found that, generally speaking, every nerve of the neck, trunk, and lower extremities is the seat of numerous and variously sized globular or oval, firm growths. Thus both pneumogastrics and their connecting and other branches carry innumerable tumors that appear to spring from the peri- and endoneurium; the large trunks contain so many small tumors in their interior that they appear as if diffusely enlarged, the larger masses giving rise to nodules in the course of the nerve (Fig. 1). In the smaller branches the relation of the tumor to the sheath is often clearly shown, the nerve running over the tumor. One of the œsophageal branches presents a globular growth as large as a walnut, which presses upon the gullet and causes a narrowing of the lumen.

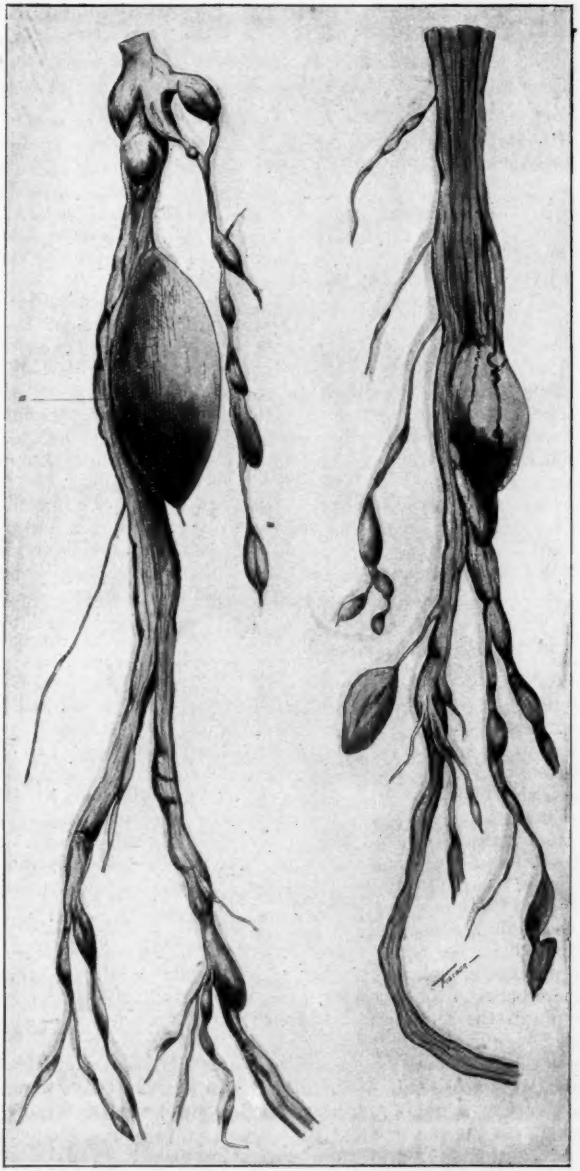
The sciatic nerves, which were hurriedly dissected out, together with the tibial nerves, present a similar well-marked diffuse nodular enlargement of the main trunks, which appear at least four times the normal size (Fig. 2); this great enlargement is likely due to the presence of tumors in the nerves, but here there also seems to be a diffuse overgrowth of the structures. The largest tumor of all is a cylindrical mass, 12 by 3.5 cm., situated loosely between the muscles on the anterior part of the right thigh and connected with a small intermuscular branch. The larger nerve trunks of the upper extremities are not dissected out, but feel markedly nodular on palpation.

The following joints were examined: The left wrist-joint, the metacarpo-phalangeal joints of the left hand, the right knee- and hip-joints. The right hip-joint is empty and the articular surfaces held in such close apposition by the thickening of the ligaments that partial ankylosis exists. The articular cartilages are thinned and somewhat roughened, but there is no distinct erosion and no marked osteophytic formations. The head of the femur is reduced in size. The knee-joint contains a small quantity of clear, viscid fluid, in which are small, grayish, turbid masses. The synovial fringes are changed into a soft, reddish, and granular tissue; surrounding the articular cartilages, which are thinned and roughened, are extensive erosions of the bone, in which lies a sort of granulation tissue that seems to be in direct connection with the red marrow that fills the medullary spaces; extensive resorption of the upper end of the tibia has taken place; the capsule and ligaments are thickened. Generally speaking, similar changes are present in joints of the hand and wrist (Fig. 3). The extensive resorption furrows around the articular bone ends are especially well marked; there is considerable clear viscid synovial fluid with gray flocculi, also soft masses of tissue on the synovial membranes, and thickenings of the capsules. There is probably some new bone at the margins of the grooves at the bone ends.

The marrow of the long bones is red.

Anatomical Diagnosis. Multiple fibromata of the cerebro-spinal and

FIG. 2.



Tumors of sciatic nerves and their branches. At *a* large tumor connected with small intermuscular nerve.

sympathetic nerves; compression of the spinal cord; polyarthritis deformans with kyphoskoliosis, ankyloses, and contractures; moist gangrene of left foot; decubitus; chronic dermatitis of left leg; inflammation of frontal sinus; chronic nephritis; marasmus.

Bacteriological Examination. Cultures made from the heart's blood, all the solid organs, and four sets of five tubes each of blood-serum inoculated with the contents of various joints; two tubes of human placental blood-serum inoculated with the same material.

Cover-slip preparations from the synovial fluids and from the synovial tissues stained in a great variety of ways do not show any micro-organisms, and all the cultures from the joints remain sterile.

A non-liquefying micrococcus, which takes Gram's stain and shows a marked tendency to arrange itself in fours, was isolated from the heart's blood and the spleen. The lungs contained the proteus vulgaris, the liver the colon bacillus; the kidneys were sterile.

Histological Examination. The various tissues were fixed in 4 per cent. formalin, Zenker's fluid, Müller's fluid, and in alcohol. Some of the bones and joints of the fingers and of the metacarpus of the left hand were placed while fresh in a mixture containing 10 parts of nitric acid and 90 parts of formalin, some in concentrated aqueous solution of picric acid, and others were decalcified in 5 per cent. solution of nitric acid in alcohol after hardening. Preparations from specimens decalcified in the formalin and nitric acid mixture, which acts rapidly and safely, show the histological structure very well indeed.

Frozen sections of the lungs show some emphysema, the lumen of the bronchi is filled with round cells and inflammatory exudate, and there is some infiltration in the surrounding air cells. In the liver there is slight infiltration in Glisson's capsule. The adrenals seem quite normal. The ovaries are normal; also the spleen and the kidney. The heart muscle has a visible cement substance, and the amount of pigment in the cells is increased. The abdominal lymph glands are normal; the inguinal lymph glands are vascular and cellular, but there are no necrotic foci.

The lining of the right frontal sinus is the seat of a marked round-cell infiltration; the covering cells show considerable mucous degeneration, and in places marked desquamation has taken place.

The yellow, irregular thickening of the serous coat of the small intestine is shown in microscopic section from embedded pieces to depend in part upon small cellular accumulations in the serous and subserous tissue and in part upon hyaline thickenings in the same places. These changes have taken place largely at the expense of the external muscular fibres, those immediately underneath the small hyaline areas being atrophic and encircled by bands of hyaline material. In the cellular area the cells are endothelioid in appearance, having deeply stained nuclei and clear bodies; they are round or oval in outline. Small cell accumulations, as well as scattered cells of similar kind, also occur here and there between the muscular fibres, especially in the outer coat. In a few places the serous coat is strewn over by a fine yellowish-brown granular pigment without any other changes being present. In some places in the small intestine the submucosa is congested and œdematous, the vascular spaces being widely dilated and the connective-tissue fibres spread apart by a finely granular or homogeneous eosin-staining material which contains some scattered leucocytes. The mucous membrane

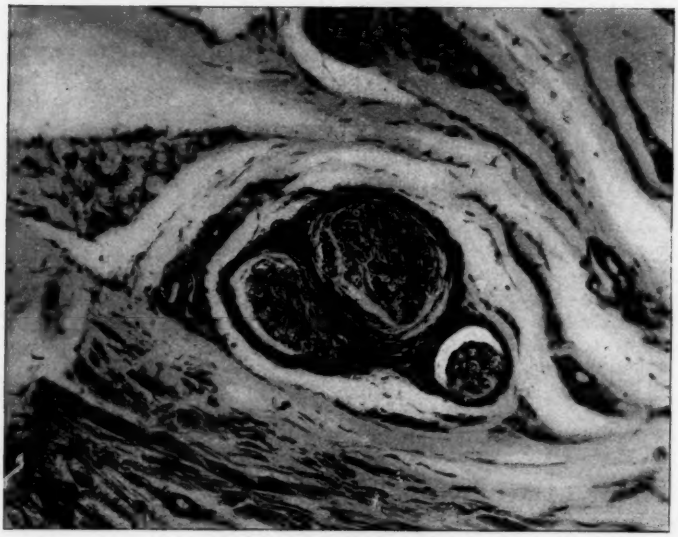
FIG. 3.

Skiagraph of left hand, showing absorption at articular ends and surface. (Courtesy of Dr. Smith, X-ray Laboratory, Presbyterian Hospital.)

is partly absent; it is cellular, and contains in many places a fine, granular, brownish pigment. Sections stained for bacteria fail to show any. The distal end of the vermiform appendix is occluded, there being no mucous membrane present. In the proximal two-thirds the lumen is very small and filled with desquamated and mucoid cells; the muscular walls are much thickened, and in the subserous coat are small areas of more or less dense cell infiltration.

Paraffin sections of the thickened area in the skin of the leg show much increase in the depth of the corium, which is quite cellular, and forms a few broad, variously shaped but mostly rounded papillary projections, covered by a quite normal epithelium, which is of varying thickness and again covered by an abnormally thick layer of horny material.

FIG. 4.



Chronic fibrous neuritis of small cutaneous nerve of left index finger. Hæmatoxylin and eosin. × 150.

The Sympathetic and Peripheral Nerves. The following description is based on the study of sections of embedded specimens stained with hæmatoxylin and eosin, Von Gieson's method, and Weigert's method for demonstrating the medullary sheaths.

The majority of the nodules observed in the mesentery are fibromata connected with nerves, and not lymph glands; frequently a small lymph node and a fibroma occur side by side; other fibromata occur upon nerves in close proximity to vessels. The fibrous tumors appear to spring from the nerve sheaths. They consist of a typical, fibrillated, more or less cellular and more or less dense connective tissue. In most

cases the fibrillar network is extremely tangled, but in some cases the fibres pursue quite parallel courses, bundles of parallel fibres crossing each other. The number of bloodvessels varies; in some places the tumors are quite vascular. The pigmented nodule in the pelvis appears to be an unusually vascular and cellular fibroma; in some places cells are especially numerous about small vessels; some quite large vessels have thin walls, and the tissue in which they occur is usually young. A few small recent hemorrhages are present; the nodule contains no ganglion cells.

FIG. 5.

Degenerated nerves in fibroma of sciatic nerve. Weigert's stain. × 100.

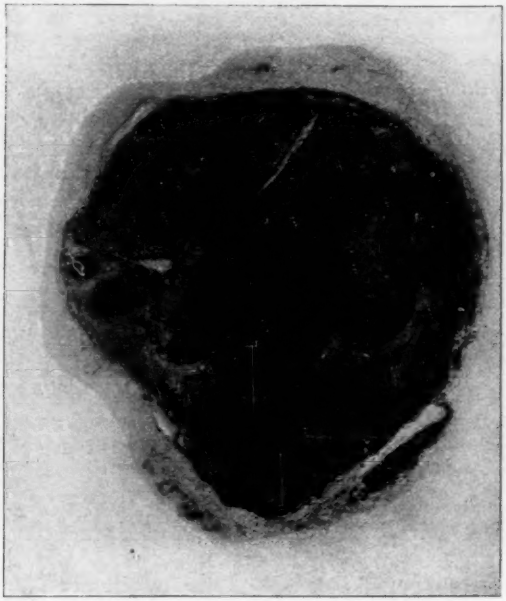
Peripheral Nerves (Figs. 4, 5, 6). In some places the large fibrous mass in the thigh is somewhat œdematous. The sciatic nerve is the seat of large and small fibrous growths, circumscribed as well as quite diffuse, the fibrous tissue being distributed throughout nerve bundles, which then present the appearance of a marked proliferative interstitial neuritis. Many of the smaller nerve bundles with increased amount of connective tissue show an unusually large number of bloodvessels. Some of the arteries in the tissue between the nerve bundles show an almost complete obliteration of the lumen by intimal thickening. In

the smaller bundles appear small vessels, whose lumen is wholly filled with nucleated cells.

Sections from the skin show many smaller and larger subcutaneous fibromata. The perineurium of the nerves of the skin is quite generally thickened and cellular, and there is evident increase in the fibrous tissue in the interior of the nerves not the seat of circumscribed tumors (Fig. 4).

Longitudinal sections of the sciatic nerves, stained by Weigert's medullary sheath method, show many intact nerve bundles and also many scattered and single, more or less degenerated nerves coursing through the intraneural fibrous nodules (Fig. 5). In some of the diffusely thickened nerves there may be but a few healthy fibres; in others the fibres are quite numerous, partly healthy, partly degenerated, and spread apart by bands and areas of fibrous tissue.

FIG. 6.



Cross section of anterior tibial nerve. Nerves in central fibromata. Weigert's stain. × 100.

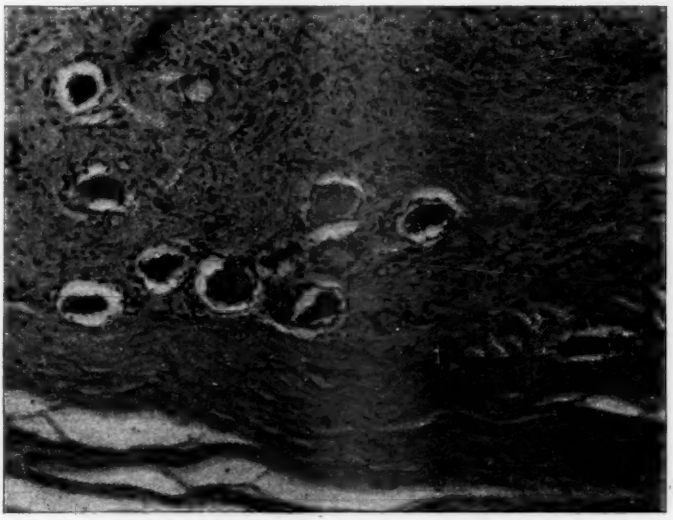
Cross sections of the anterior tibial as well as of other nerves, stained in the same way, show also some healthy fibres in circumscribed, often central, tumor nodules, arranged around which are nerve bundles, in some of which are markedly degenerated districts (Fig. 6).

Many tumors give in the sections a quite decided reaction with Weigert's stain for elastic fibres, but accurate study of the distribution and amount of elastic tissue has not been made.

In none of the tumors were there any evidences of development of sarcoma.

Sections from the central part of the tumor corresponding to the fourth right cervical spinal ganglion show a diffuse fibromatous proliferation of the ganglion, the ganglion cells are pressed far apart by the fibrous tissue, and occur singly and in small groups (Figs. 7 and 8). The ganglion cells show more or less shrinking of the body, occasional vacuoles in the protoplasm, and apparently the capsules are rather more cellular than in some of the healthy ganglions examined. When the ganglion cells occur singly they generally have an oblong and narrow form, and lie parallel with the bundles or strands of fibrous tissue. A few ganglion cells have been reduced to shapeless remnants without nuclei.

FIG. 7.



Section of spinal ganglion involved in fibroma. Hæmatoxylin and eosin. × 150.

The hardened brain, when cut into thin transverse and longitudinal slices, shows only normal appearances.

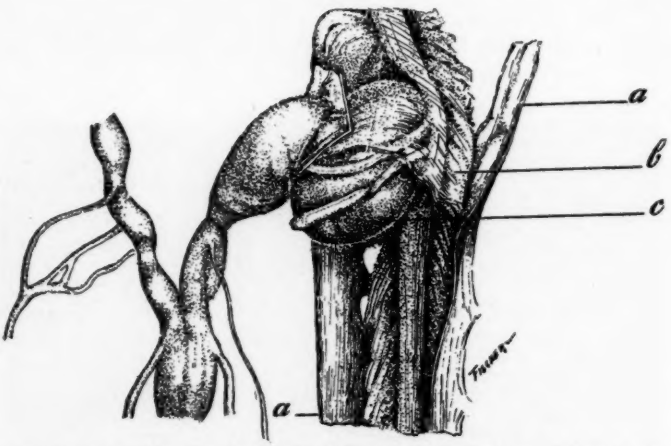
Examination of the formalin-hardened spinal cord shows that the gross changes due to the compression extend over a distanee of 3½ cm. where the cord is greatly flattened (Fig. 8). The cord is thinnest in the centre of this part, and here it measures 1 cm. in breadth and 2 to 3 mm. in thickness. It has a concavo-convex appearance, the margins of the compressed part being the thinnest. The pia-arachnoid cannot be readily loosened from the surface of the flattened portion, which seems firmer than the cord ordinarily is. At the greatest change the cut surface does not present any recognizable normal outline. As one approaches the limits of the compressed part, it can be readily

seen that the compression has been applied first upon the posterior-lateral aspect of the left half, resulting in the disturbance first of the right posterior horn and posterior columns, and these are the parts which lie immediately subjacent to the subdural fibromata (Fig. 10).

There are no cavities in the compressed part, and the central canal is not dilated.

The changes in the cord and medulla were studied by Mr. Martin H. Fischer, with the following result:

FIG. 8.



Compression of cord by intraspinal fibroma of fourth right cervical nerve. *a*, dura; *b*, pia, with compressed cord underneath; *c*, fibroma of the nerve. (Drawn from behind.)

Section through Middle of Medulla (Weigert's stain).

Distinct degenerative changes exist in the funiculus gracilis and the funiculus cuneatus. The restiform bodies show large numbers of degenerated fibres. The anterior pyramids show a general lightening. The evidences of degeneration are most marked in the central and peripheral portions of these tracts.

Section at Junction of Medulla and Spinal Cord.

The column of Goll is represented by a triangular lightened area lying close against the posterior median fissure. The apex rests against the gray commissure. At the periphery of this degenerated tract scarcely any nerve fibres can be found. The peripheral portion of the column of Burdach has suffered more severely, the most central part having suffered but little. The direct cerebellar, the lateral descending and ascending, and the direct pyramidal tracts show severe grades of degeneration. The process is most marked in the peripheral portions of the cord. The decussating fibres of the pyramids seen at this level are well stained in Weigert's preparations, though some degenerative changes can be seen.

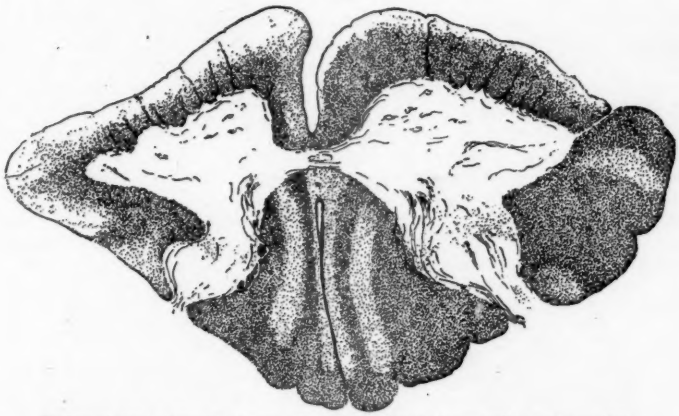
The areas of degeneration are, in the main, symmetrical; still, those of the left half of the cord are of a severer type than those of the right.

Section above the Tumor, Opposite the Fourth Cervical Vertebra (Fig. 9).

The entire left side of the cord has undergone a diminution in size. The columns of Goll and Burdach each show well-defined tracts of degeneration. The direct cerebellar, the crossed pyramidal, the antero-lateral ascending and descending, and the direct pyramidal tracts of the left side show scarcely any healthy nerve fibres. The crossed pyramidal tract of the same side shows a general lightening. On the right side a light rim of total degeneration marks the direct pyramidal and the antero-lateral ascending and descending cerebellar tracts. The entire anterior column of this side seems to have suffered a contraction. The tract of Lissauer shows a fairly well-marked degeneration on the left, but only a slight lightening on the right side.

The anterior and posterior horns of the left side show a diminution in size.

FIG. 9.



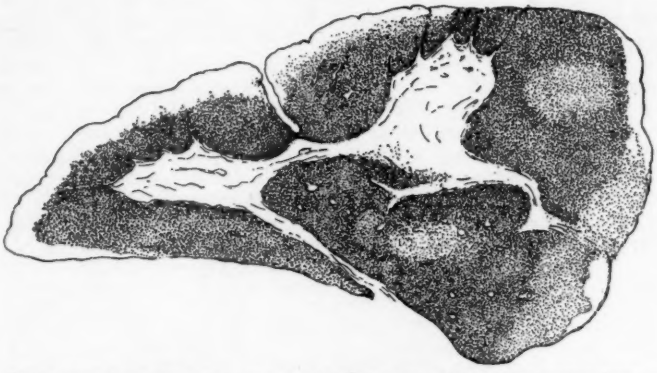
Section of cord above the tumor. For description of degenerated areas, see text. Weigert's method.

Section Opposite the Tumor (Fig. 10).

A compression of the cord substance has occurred to the right and forward. The compression involves the left lateral and posterior and the right posterior columns. The posterior median fissure has been obliterated. A homogeneous unstained rim marks the position of the direct cerebellar, antero-lateral ascending and descending, and direct pyramidal tracts on the left side. The crossed pyramidal tract has suffered partial obliteration, and is perhaps represented by a lightened area of fibres at the posterior portion of the anterior horn. A marked degeneration is also noted in the direct pyramidal tract of the right side, with a questionable lightening of the crossed pyramidal tract. A narrow, well-defined area of degeneration is noted in the direct cerebellar

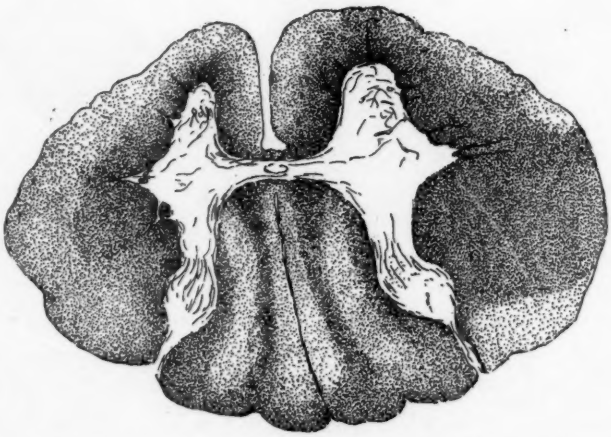
tract of this same side. The two posterior columns have been fused into one through the obliteration of the median fissure. In the central portion of this is seen an area of lightening, as also just internal to the apex of the right posterior horn, which may correspond to the degen-

FIG. 10.



Section of cord opposite tumor. For detailed description, see text. The section shows the deformity of the cord. Weigert's stain.

FIG. 11.



Section of cord opposite dorsal vertebra. For detailed description, see text. Weigert's method.

erations observed above in the columns of Goll and Burdach. The left anterior horn is markedly increased in size. It is flattened antero-posteriorly, and the posterior horn proceeds from it backward and inward as a narrow unstained band.

Section Below the Tumor, Opposite the Third Dorsal Vertebra (Fig. 11).

The columns of Goll and Burdach show a general lightening. A well-marked lightening has also occurred in the left lateral column of the cord, involving the crossed pyramidal, the direct cerebellar, and the antero-lateral ascending and descending tracts. The crossed pyramidal tract is involved only in its external portion. In the anterior columns the direct pyramidal tracts show marked lightening on both sides of the cord. The antero-lateral ground substance on both sides shows no change. A slight lightening has occurred in the antero-lateral ascending and descending tracts of the right side. Just external to the apex of the posterior horns small areas of degeneration are seen which correspond to the tracts of Lissauer. The anterior horn of the tumor side shows a diminution in size, having lost the rounded form possessed by the horn of the other side and assumed a more pyramidal shape. The posterior horns are also thinner than on the right side.

Section from Mid-lumbar Region.

The areas of degeneration correspond in the main to those described in the sections from the mid-dorsal region. A larger number of healthy fibres are found just around the gray matter in the antero-lateral ground bundle on both sides. The degenerated tracts lie more at the periphery of the cord, and are rather indistinctly differentiated from each other.

The nerve fibres in the various localities show varying degrees of degeneration; many of the fibres are shrunken; others are much swollen, have lost their rounded form and become more or less oval and elliptical in shape. As a rule, the areas which show the greatest number of small, contracted fibres show the greatest increase in connective tissue (Von Gieson's stain).

The myelin sheaths of the swollen fibres stain palely with Weigert's method. The neuraxons are more or less eccentrically located; in some localities the neuraxons have been entirely lost, and only drops of myelin are left to represent nerve fibres.

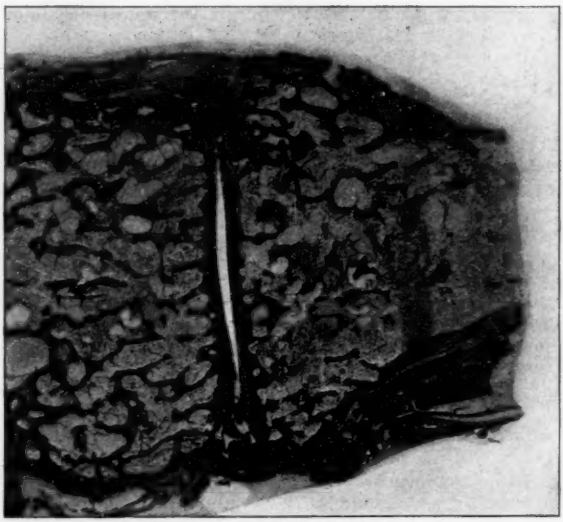
The rule that the localities which show the greatest number of small fibres show the largest amount of connective tissue increase is not an absolute one, as the anterior and lateral columns of the cord on the left side just opposite the tumor show a great number of swollen nerve fibres. It would seem from this that as the tumor grew the increasing pressure upon the cord caused more and more fibres to degenerate, so that we have fibres in all portions of the cord in all stages of degeneration.

Marchi's method shows but few black-stained fibres; these are seen most numerously in the peripheral regions below the seat of the compression, and more especially in the posterior portions of the cord. This reaction would go to indicate that while the majority of the nerve fibres represent old stages of degeneration demonstrable by Weigert's method, others are of more recent date, and so give the Marchi reaction.

Joints, Bones, Bone-marrow. The marrow of the femur is quite congested and there is considerable focal cell accumulations. Among the cells are recognized (hæmatoxylin and eosin) quite a number of richly granular eosinophiles, with small round nuclei, occasional megalocytes, and the ordinary narrow cells.

The joints of the left hand were the ones principally studied (Figs. 12, 13, and 14). The surfaces of the articular cartilages may be somewhat uneven, and just below it is a strip the matrix of which is a little fibrillated and split up, the cartilage cells being largely absent. The matrix of the principal part of the cartilage is quite normal, but there is usually more or less proliferation of the cartilage cells, leading at times to the accumulation of from four to eight or twelve cells in one capsule; the cartilaginous matrix is, moreover, rather granular and somewhat split up. In all cases the deeper layers of the cartilages are of a peculiar finely granular appearance and light bluish color (hæma-

FIG. 12.

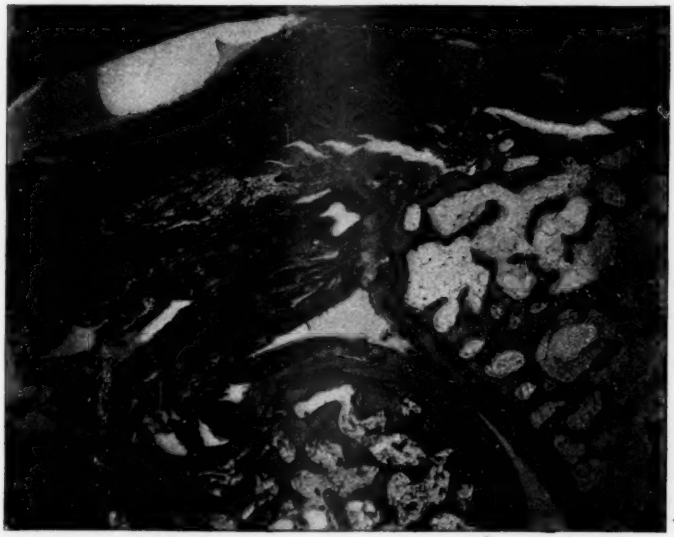


Section through a metacarpo-phalangeal joint of the left hand; shows the excavations at the margins of the articular cartilages filled with cellular connective tissue. The bone is porotic. Note the distinct junction between the cartilage and the bone. × 75.

toxylin); this zone is of a wavy outline (Figs. 12 and 13); at the margin of the more healthy cartilage there is a deeper blue color, as if a cloud of blue dust granules had been swept up to this point. The cartilage cells in this zone are largely absent or the nuclei fail to stain, so that the capsules appear empty; in some instances the capsular space contains a few small chromatin particles. This zone is sharply demarcated from the underlying bone. At the peripheral margins of the articular cartilages a vascular and cellular fibrous tissue more or less directly connected with the synovial fringes has replaced part of the cartilage and fills the excavation which has formed in the bone proper at these points, extending sometimes for some little distance into the subchondral medullary spaces as well as creeping along over the articu-

lar surface of the cartilage, which now presents an escalloped appearance (Figs. 12 and 13). Generally the line of junction between the cartilage and fibrous tissue is sharp and abrupt, but islands of connective tissue occasionally appear in the interior of the cartilage as if formed by metaplasia, the matrix of the cartilage becoming fibrillated and the cells liberated from the lacunæ, assuming the rôle of cells in fibrous tissue. The absorption of the bone has occurred principally at the margins of the articular cartilages, which often form overhanging shelvings over the fibrous tissue. There are no osteoclasts seen at the margins of the

FIG. 13.

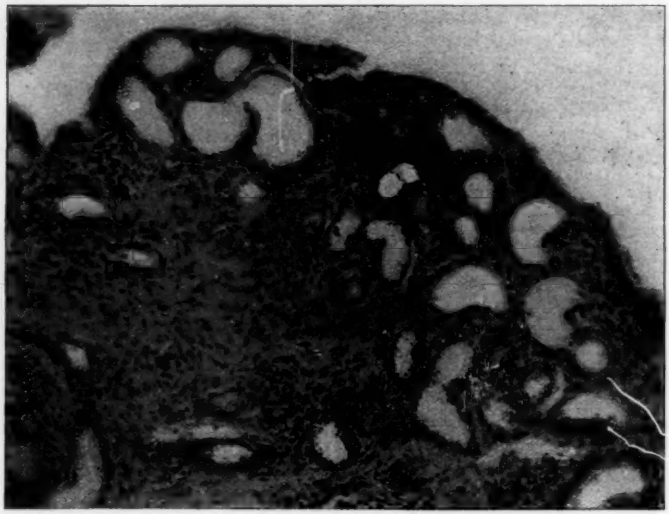


Part of Fig. 12, more highly magnified. Note replacement of cartilage at the margin by cellular connective tissue, which is creeping over the articular surface. See also wavy line at junction of bone and cartilage. × 200.

erosions in the bone. The medullary tissue is principally fatty; occasionally small focal cell-accumulations occur. At times medullary tissue has formed in the deeper layers of the cartilages. The synovial membrane where it turns over the articular cartilage is the seat of delicate and branching villous outgrowths which contain very many rather large arteries and other vessels, the stroma between being richly infiltrated with small round and oval nuclei, the whole presenting an appearance not unlike an angioma (Fig. 14). At other times the cells are larger, younger, and the vessels provided with thinner walls, so that the tissue looks like a recent granulation tissue. At some distance away from the joints there is more or less well-marked periosteal proliferation in the form of osteoid tissue.[1]

[1] These interesting changes in the bones and joints are reserved for a more complete consideration in the future.

FIG. 14.



Section of villous outgrowth of synovial membrane, showing the cellular and vascular structure. × 200.

GENERAL CONSIDERATIONS (Dr. Preble). The case is an extreme degree of a very common condition, that of generalized neurofibromatosis, but has more than ordinary interest because it presents certain features which are rare, if not unique —the joint changes and the trophic changes in the skin.

Cases of fibrous tumors growing from the fibrous portions of the peripheral nerves have been recorded in medical literature since 1793, when Tilesius (quoted by Modrzejewski) reported the first case. No correct appreciation of their true nature, however, was suggested until 1847, when Brüch suggested that these tumors were due to polypoid growth of the neurilemma. Rokitansky shared this opinion. In 1880, Prudden collected forty-one examples of this condition. In 1882, Recklinghausen, by careful study of cases, showed that the fibroid tumors of the skin, the fibroma molluscum of Virchow, arose from the fibrous portions of the peripheral nerves. Since then these cases have attracted much interest and study, so that to-day there is a vast literature and a great number of cases recorded. When to the cases frankly of this nature we add the cases of conditions probably exactly similar, so far as their pathology and pathogenesis are concerned, though different in their clinical manifestations, the number becomes very large

indeed. We should include many cases of elephantiasis and many cases of pigmented and linear nævi. The number could be easily still further increased, for lesser degrees of this condition of neurofibromatosis are so common that one could readily collect hundreds of examples in a few weeks.

Etiology. A very large number of the cases are congenital, but one cannot agree with Bruns, who says they are always congenital. There are many examples in which the first tumor has appeared some years after birth, as is illustrated by the case recorded above and by those of Generisch, in which the tumor appeared at eight, Lahman at twenty-three, Berggrün at ten, Hitchcock at nineteen, and many others.

In some cases, while no tumors are present at birth, there are congenital pigment flecks, and later the tumors appear. The case of Feindel and Oppenheim is an example of this, as is also the case of Spillmann and Etienne.

Not only are many of the tumors congenital, but in many cases they are found in other members of the same family: Herczel, mother and grandmother; Virchow, grandfather, father, and brother; Hecker, grandmother and parent; Czerny, grandfather, greatuncle, uncle, and mother; Bruns, two brothers; Hitchcock, mother and two sisters. Many other examples might be quoted, but no instance of extension through more than three generations has been found.

Some authors (Landowski and Feindel) believe, when the tumors appear late in life, that some exciting cause, such as bad hygienic surroundings, overwork, cold, trauma, or infection has been at work upon one who has a congenital predisposition to these tumors, and the tumors have resulted. The first case of Spillmann and Etienne may be an example of this. This patient, a male adult, had had pigment spots on the skin for many years, when, after a fall which stretched the sciatic nerve, tumors appeared scattered over the skin. Most authors believe, with Feindel, that there is during the development of the embryo some disturbance of the tissues derived from the ectoderm. If the disturbance is marked the effects in the way of skin tumors, tumors of the nerves and pigmented areas appear at birth. If the disturbance is less there is no sign at birth, and only later, under the influence of some incidental cause, do the results appear.

Recklinghausen, Etienne and others have suggested that the linear nævi which show such manifest relations to the peripheral nerve are due to an intra-uterine neuritis or myelitis, which so lessens the control of the nutrition of the skin that certain elements hypertrophy. Such fœtal neuritis might be due to some infection or intoxication from the mother. These linear nævi are seen frequently with skin and nerve fibromata, and it seems likely that they are due to the same underlying processes.

Other authors, notably Esmarch and Kulenkampf, suggest that there

is a fibroid diathesis which is the cause of the neurofibromatosis; others suggest that it may be an intoxication, like Addison's disease or myxœdema. Still others suggest a specific infection, which, like lepra, causes pigmentation of the skin, and tumors of the nerves and skin.

Symptomatology. Following the example of Landowski, Feindel and Oppenheim, Briquet and others, we can conveniently divide the symptoms into four groups: tumors of the skin, tumors of the nerves, pigmentation of the skin, and functional disturbances. A completely developed case shows symptoms from all four groups; but there is a much larger number of cases which are incomplete, showing symptoms from three, two, or even one group only. The case reported above showed no areas of pigmentation, but symptoms from each of the other groups were present. The two groups most frequently absent from the clinical picture of these cases reported are the pigmentations and functional disturbances. I am inclined, however, to believe that mention of the pigmentation is frequently omitted from the history sheets because of the apparent insignificance. If one takes into consideration the very numerous, incompletely developed cases seen almost daily in the clinic, one must conclude that the two symptoms commonly present are skin tumors and pigmentation, while the tumors of the nerves, and especially functional disturbances, are the exceptions.

Skin Tumors. The number of tumors varies from one or two to many thousands. In the case reported above there were many hundreds of tumors in the skin, scattered irregularly over the body. Modrzejewski reports one case with 3000 tumors. Virchow, in his book on tumors, has a picture of a patient who must have had thousands of these tumors. Lesser, in his text-book upon diseases of the skin, figures a patient who presents many hundreds of the ordinary forms of the fibroma of the skin and nerves, and also the larger form, the molluscum fibrosum, *Rankenneurom*, or elephantiasis neuromatodes.

The tumors vary in size from a pinhead to a large egg. They may be sessile or pedunculated, tending to become the latter as they increase in size. The skin covering them may be perfectly normal, but with many of the tumors the skin is thin, relaxed, and thrown into fine, irregular folds, looking as if it had been stretched for some time and then relaxed. Ordinarily the skin covering these tumors contains less than the normal amount of pigment, but it may be pigmented. Sometimes the skin grows large comedones; but these cases, as will be shown later, are probably not neurofibromata. The tumors are usually soft, but may be hard and dense. They are usually neither tender nor painful. They cause no disturbance except mechanically from their site or size.

In addition to these tumors, which are the common form, there are others of exactly the same nature, described under various names:

fibroma molluscum, *Rankenneurom*, and elephantiasis neuromatodes. The resulting tumors are often very large, and may take the shape of fairly circumscribed tumors or of irregular thick folds, and laps of skin, such as are seen in the other forms of elephantiasis. The skin covering these folds may be unaltered until the mass becomes very large, when it tends to become inflamed. The skin is much more frequently pigmented than with the ordinary fibromata, and in some cases may be covered by a growth of hair. The *Rankenneurom* is often seen in combination with the ordinary tumors, as is illustrated by the cases shown by Lesser, Herczel, Czerny, Kriege, Bruns and others, but may occur without them, but with pigmentation, as reported by Bruns; or we may find skin tumors, nerve tumors, and *Rankenneurom* combined in the same patient, as reported by Kriege.

These large tumors cause no disturbance except such as results mechanically, and cannot be distinguished clinically from other forms of elephantiasis except by the presence of the commoner manifestation of generalized neurofibromatosis.

Nerve Tumors. These may occur alone—*i. e.*, without skin tumors—as is illustrated by the cases of Lahmann, Kriege, Keen, Spiller and others; but, as a rule, when multiple nerves are involved there are tumors of the skin also. Picque collected fifty cases of plexiform neuromas, in eighteen of which it is stated that there were cutaneous neurofibromata. The relative importance of the nerve and skin tumors varies in the individual cases. For example, in the case above, the tumors of the nerves are much more numerous and important. This is also true in the very extreme case reported by Berggrün.

The tumors vary in size from those which are too small to be palpated to the size of the large tumor in the right thigh—12 by 3.5 centimetres, or even larger. The tumors are round, oval, or fusiform, not attached to the surrounding parts, so that motion from side to side is permitted, but longitudinal motion in either direction is prevented by the want of elasticity of the nerve. Usually there is more than one tumor on a nerve, thus presenting an arrangement like a string of beads. The tumors may develop along the trunk of a nerve or along its branches. When the latter is true irregular convoluted masses result, which feel not unlike a varicocele, except for their density. The tumors, as a rule, are firm, but in some cases they are cystic. They are usually neither tender nor painful, but may be. The patient reported above had one tumor in the left side of the neck, which was tender and painful, although post-mortem it differed in no way from the other tumors found.

The tumors may affect a few nerves, or many, and there is no nerve in the body which may not be affected. The spinal, cranial, and sympathetic nerves are all found involved in many cases. As a rule, the involvement of the spinal and cranial nerves does not extend into the

cranial cavity, but there are numerous exceptions. For example, the case of Generisch and Berggrün, in which both cranial and spinal nerves were enlarged into the cranial and spinal cavities, and in the case of Czerny and the one above in which the enlargement of the spinal nerves extended within the dura, but the cranial nerves within the cavity escaped.

Pigmentation occurs in a variety of forms, colors, and arrangements. In many cases the only portions of the skin which are pigmented are those covering the tumors. In others the pigment occurs as flat or but slightly elevated areas, varying in size from a pinhead to a large plaque. The pigment flecks are usually most numerous over the covered part, the trunks, the thighs, and arms, while the face, arms, and legs usually escape. Generally there is no sign of an arrangement of the pigmented areas, but in the case reported by Dupin and Dieulafe many of the pigmented areas were elliptical in shape, with a tendency for the long axis of the ellipse to lie parallel to the long axis of the limb. Feindel and Oppenheim note in their second case that some of the pigmented areas follow the course of the nerves.

The color of these areas varies from light brown, café au lait, to dark brown. Usually there are no hairs in the pigment flecks, but in some cases, as in those of Marie and Bernard, Delore and Bonne, they are found. Briquet includes hairy and vascular nævi in his list of symptoms of this condition. In a case reported by Depaul and Verneuil, a tumor of the neck near the scalp was covered by thick, coarse hair, which differed from that covering the scalp. Below this tumor were, under thick, brownish skin, corresponding exactly to the distribution of the cervical plexus, nodular strings like a varicocele.

Functional Disturbances. One might expect that the tumors of the nerve would cause marked disturbances in their functions; but, as a matter of fact, even slight disturbances are rare. The commonest symptom in this group is pain, which may be spontaneous or appear on pressure. The pain may be in the tumor, or radiate to the area supplied by the nerve affected. Actual disturbances of function are less common, and may be sensory or motor, and be either of the nature of excitation or paralysis. Paræsthesia, hyperæsthesia, anæsthesia, cramps, tonic convulsions, weakness, and paralysis are all seen. A small number of cases suffer from arthralgia. In a few cases symptoms from involvement of the vagus have been seen: dysphagia, hoarseness, dyspnœa, palpitation, and tachycardia (Generisch).

Briquet saw vomiting and cramps in the abdomen in one case.

The case reported by Berggrün shows more marked disturbances than any case recorded, but in his case the enlargements on the cranial nerves extended to the cranial cavity, and many of the symptoms, such as paralysis of various of the motor nerves of the eyes and the optic

neuritis were due to the intracranial tumors rather than to the fibromatosis of the nerves. Those few cases in which there has been paralysis of the bladder and rectum, atrophy of certain muscles, were probably cases in which the cord was compressed by tumors in the spinal canal. Extension into the spinal canal does not always cause symptoms, even when there is considerable pressure of the cord, as is illustrated by the cases of Generisch and Czerny and the case above.

Many of the patients are of a low order of intelligence, apathetic, languid, and stupid; but this is scarcely the rule, as some of the French authors consider it to be.

No descriptions of trophic disturbances have been found in any of the reports; there is, indeed, no mention made of them except by Landowski, who says they are very rare, and von Büngner, who says they are usually absent.

The patient J. S. had for some years on the posterior aspect of one thigh an ulcer which looked in all ways like the perforating ulcers so often seen on the feet of tabetic patients, and resisted treatment in the same way. This was looked upon clinically as a perforating ulcer of trophic origin. The gangrene of one foot coming on spontaneously was regarded as of the same nature.

The joint changes in this patient might also be regarded as due to nerve influence. Their painless course and the great resultant deformity, together with the symmetry of the involvement in a patient whose peripheral nerves were so greatly altered, naturally suggested this idea. The distribution, deformity, and involvement of the spine were such as are seen in well-marked cases of arthritis deformans. In reviewing the literature no mention of joint changes was found except in the report by Berggrün and that by Dupin and Dieulafe. In Berggrün's case both hips and knees were strongly flexed, but could be moved passively, although the amount of motion in the hips was slight. The ends of the right femur and tibia were markedly thickened. At the autopsy the head of the left femur was out of the socket, the teres ligament was relaxed, the joint cavity was small and filled with loose, red tissue, and the head of the femur was flattened. Dupin's and Dieulafe's case suffered from rhachitis and repeated attacks of acute articular rheumatism when young, and the bone and joint changes may easily be the results of the early illnesses. The lower epiphyses of the forearm are enlarged, the first phalanges are flexed on the metacarpal bones, the third phalanges are extended, the phalanges are subluxated because of the relaxation of the ligaments, and dorsal scoliosis and double genu valgum are the changes noted. These are the only recorded cases of joint changes in the course of neurofibromatosis, and inasmuch as the disease in its milder forms is very common, and not very rare, even in its severe forms, I believe, we must give up the very fascinating idea

that there is some intimate relation between the two conditions and regard the combination as merely accidental.

The course of the disease is slowly progressive, and only in the severest cases causes trouble and danger. Rarely we find exceptions to this slow course, as is illustrated by the patient of Berggrün, who died at the age of eleven, directly from the effects of the neurofibromatosis. Many of the patients die from some intercurrent disease; others develop a gradually increasing anæmia and cachexia, and die from exhaustion; still others die from the effects of the accidental location of some tumor; while some die from sarcomatous degeneration of the fibrous tumor. This last is by no means infrequent, so that numerous cases have been recorded. Garré collects seventeen cases of sarcomatous degeneration. The cause of this change in the nature of the tumor is unknown, but in some cases it seems to have been some trauma.

The *prognosis* in the milder cases is good, but in the severer forms must be conservative because of the danger of a tumor developing at some spot where it can mechanically cause serious disturbances, and because a tumor may at any moment become malignant.

Diagnosis. This is usually easy because of the marked and peculiar nature of the symptoms. Lahman has drawn attention to the fact that multiple fibromata of the skin may arise from the fibrous tissue of other parts of the skin than the nerves, and proposes dividing these fibromata into four classes according as they arise from the fibrous tissue of the nerves, the vessels, the glands, or from several of these. The differentiation of these may require microscopical examination unless there are deeper tumors having the character, location, and arrangement of tumors of the nerves.

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THE RELATION OF CHOLELITHIASIS TO DISEASE OF THE PANCREAS AND TO FAT NECROSIS.

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Since the common bile-duct and the main duct of the pancreas unite in the diverticulum of Vater before reaching the lumen of the duodenum, changes occurring in one may produce pathological conditions in the other and consequent lesions of the liver or pancreas. The object of the present paper is to direct attention to the occurrence of pancreatic disease as a complication of gallstone colic, to the method of its occurrence, and to the nature of the lesions which ensue.

The association of the two conditions has been incidentally mentioned by a number of writers. In the several recently published monographs on diseases of the pancreas reference to it is made. Kôrte,[1] writing on the surgery of the pancreas, notes that diseases of the bile passages, especially cholelithiasis, are frequently associated with lesions of the pancreas, and thinks it is probable that inflammation can extend from the bile-duct to the gland. Oser[2] thinks that catarrhal inflammation may extend from the gall passages to the pancreatic duct, producing obstruction of the secretion and its consequences; the pancreatic duct may be occluded by a stone lodged in the common duct near its orifice. Lancereaux[3] mentions the possibility that a gallstone lodged in the common duct at the level of the diverticulum of Vater may occlude the pancreatic duct and produce conditions favorable to the penetration of micro-organisms into the pancreas. Ebstein[4] has recently reported a case of diabetes associated with attacks of abdominal pain. Seeking an explanation of the obscure symptoms, he discusses the interrelation of pancreatic and hepatic disease. He recalls the fact that the ductus communis choledochus and the duct of Wirsung enter the intestine side by side, so that a gallstone or a pancreatic calculus lodged near the orifice of either duct might occlude both.

Gallstones are not infrequently accompanied by inflammatory changes in various parts of the bile passages, and it is conceivable that secondary lesions of the pancreas may follow. The inflammatory process whose immediate cause is the presence and development of micro-organisms may extend along the common duct, to the diverticulum of Vater, be transmitted to the main pancreatic duct, and thence to the parenchyma of the organ. Biliary calculi may favor the occurrence of pancreatic lesions by another method. Lodged near the orifice of the common duct they may compress the neighboring duct of Wirsung and obstruct the outflow of the pancreatic juice. When the duct is wholly or partially occluded one may readily believe that, though the secretion of the gland cannot enter the intestine, micro-organisms may make their way from the duodenum into the dammed-back secretion, and, multiplying, produce inflammatory changes in the gland. The obstructed secretion, moreover, may be forced backward into the gland parenchyma, and, even though micro-organisms have not entered, may produce injurious effects upon the substance of the organ.

When the common bile-duct is occluded the secretion of the liver forced back upon the organ finds its way into the interstitial tissue, whence it is transmitted to more distant parts by way of the lymphatics

[1] Chirurg. Krankh. des Pankreas. Deutsche Chirurgie, Stuttgart, 1898.
[2] Die Erkrank. des Pankreas. Nothnagel's Spec. Path. u. Ther., Vienna, 1898, vol. xvii.
[3] Traite des malades des foie et des pancréas, Paris, 1899.
[4] Zeitsch. f. klin. Med., 1900, vol. xl. p. 181.

and reaches the blood through the thoracic duct. The bile-pigments stain the tissues a conspicuous yellow color and indicate the presence of retained products of secretion. The pancreatic juice, on the contrary, containing no pigment, does not carry an index of its presence. When the pancreatic duct is occluded, do we find any evidence that the secretion has penetrated the surrounding tissue and there produced deleterious effects?

Disseminated fat necrosis was first accurately described by Balser in 1882. Not infrequently one finds studding the fat in the immediate neighborhood of the pancreas opaque, white areas, varying in size. Less frequently similar opaque foci are found in the omentum, mesentery, and subperitoneal fat of the abdominal walls; rarely in the pericardial and subcutaneous fat. Microscopical examination shows that the fat cells in these areas are necrotic, and the micro-chemical studies of Langenhans[1] have demonstrated that the necrosis is associated with the splitting of the contained fat into its fatty acid and glycerin. The former, being insoluble, remains in the cell, and subsequently unites with calcium salts, while the soluble glycerin is absorbed and carried away. Various lesions of the pancreas have been found associated with fat necrosis, notably hemorrhagic and gangrenous pancreatitis, more rarely other changes. The investigations of Langenhans, Hildebrand and Dettmer, Milisch, Williams, Flexner, Katz and Winkler, and Opie have shown that the active factor in the production of necrosis of the fat cell and disintegration of the fat is the fat-splitting ferment of the pancreatic juice, which as the result of some lesion of the gland has made its way into the surrounding tissue and come in contact with the fat cells.

In a series of experiments recently reported[2] I have shown that ligation of both pancreatic ducts in the cat causes very wide-spread fat necrosis, involving almost the entire abdominal fat, at times the subcutaneous and pericardial fat, and reproduces the wide distribution occasionally observed in human beings and in animals. If the ducts are ligated and pilocarpin is subsequently administered to the animal in order to stimulate the secretion of the pancreas, the dissemination of the pancreatic secretion is much hastened and very wide-spread focal fat necrosis rapidly occurs. The experiments show that when the outflow of the pancreatic juice is obstructed the products of secretion, dammed back upon the gland, penetrate into the tissue surrounding it and produce alterations which serve as an indication of their presence. The pancreatic lesions which in human cases are associated with necrosis of fat are such as permit the penetration of the secretion into the tissue.

[1] Virchow's Arch., 1890, vol. cxxii. p. 252.

[2] Contributions to the Science of Medicine. Dedicated to Wm. H. Welch, M. ., Baltimore, 1900, p. 859.

Fat necrosis may be regarded as an index of pancreatic disease, and is, therefore, an important factor in the study of lesions affecting the organ. The condition is a consequence of pancreatic lesions much as jaundice is a symptom of hepatic disease, and is not, therefore, an independent morbid entity.

In recording the experiments referred to above I have taken occasion to point out that obstruction of the pancreatic duct in human cases is not infrequently associated with disseminated fat necrosis. The condition occasionally follows occlusion of the duct by calculi or by carcinoma. Several cases are cited where gallstones were found lodged in the common bile-duct near its orifice in such a position that they might at the same time occlude the pancreatic duct.

The anatomical relations of the common bile-duct and the duct of Wirsung are well known. The common bile-duct descends toward the duodenum alongside the head of the pancreas, occasionally embedded in its substance, and comes in contact with the duct of Wirsung, beside which it lies for a short but variable distance before entering the wall of the intestine. The two ducts penetrate side by side the coats of the duodenum, and after passing obliquely a distance of about 2 cm. and causing a papilla-like elevation of the mucous membrane, unite to form a short cavity—the diverticulum of Vater. Near its termination, at a point where the two ducts are in contact, the common duct becomes constricted, and it is here that a foreign body passing downward tends to lodge.

Of considerable importance is the fact that occlusion of the main pancreatic duct does not necessarily cause complete obstruction to the outflow of the pancreatic secretion. The duct of Wirsung communicates within the gland with the accessory pancreatic duct or duct of Santorini, which opens into the duodenum at a point 2.5 to 3.5 cm. nearer the stomach. Schirmer[1] has studied the relation of the two ducts in a large number of human cases, and has found that it presents much variation. Among 104 bodies he found that in sixty-five instances the accessory pancreatic duct anastomosed with the larger within the organ and opened by a separate orifice into the duodenum. In one instance three ducts entered the duodenum. In four cases the common bile-duct alone opened into the diverticulum of Vater, while the single pancreatic duct entered the duodenum at a point corresponding to the usual site of the orifice of the accessory duct. In thirty-four instances the smaller duct did not anastomose with the larger or communicate with the duodenum. In the latter case alone would it be possible for a stone lodged in the diverticulum of Vater or in the common duct, where it comes in contact with the pancreatic duct, to obstruct completely the outflow of the

[1] Inaug. Diss., Basel, 1893.

pancreatic secretion. In only slightly more than one-third of the cases where gallstones lodge near the orifice of the common duct should one expect to find changes in the pancreas referable to duct obstruction.

Opportunity has recently been afforded for the study of a case in which lesions of the pancreas were associated with the impaction of a stone near the orifice of the common duct. In view of the obscurity in which the etiology of pancreatic lesions is still involved, it may be of interest to record the case and to compare it with others in which a similar etiological factor is more or less clearly demonstrable.

L. F., male, aged forty-seven years. The patient was admitted to the Johns Hopkins Hospital, service of Dr. Osler, complaining of abdominal pain and fever. His family history is unimportant. With the exception of rather frequent attacks of indigestion, characterized by pain after eating, distention, and rarely nausea and vomiting, he has enjoyed good health. He does not use alcohol in any form. Six months before his present illness he suffered from an attack of jaundice, lasting about three weeks, and accompanied by abdominal pain and some fever. His stools at this time were somewhat clay-colored. At the end of three weeks he felt perfectly well.

The present illness began eighteen days before admission to the hospital, when about 9 P.M. the patient was suddenly seized with violent nausea and vomiting, accompanied by intense cramp-like pain in the abdomen. The vomiting continued during the first night, and has since only occasionally recurred. The abdominal pain, which was not localized, remained severe during four or five days, and at times there were symptoms of collapse. The abdomen was distended and the bowels were constipated until the fifth day, when, with the aid of a purgative, movement occurred. The stool was normal in color. On the third day elevation of temperature to 101.5° F. was noted. About the seventh day tenderness and slight swelling were noticed in the right hypochondriac region. Since this time the patient has had an irregular temperature (100° to 103° F.), with several chills; the pulse, 100 to 120 a minute, has been at times dicrotic. Since the first few days the abdominal pain and tenderness have not been severe, but distention of the abdomen has gradually increased. Jaundice has not been noted

Physical Examination (note by Dr. Futcher). The patient is a large-framed man. The complexion is somewhat sallow and the conjunctivæ have a slightly yellow cast. The pulse is of fairly good volume, regular in force and rhythm. Examination of the heart and lungs is negative. On inspection of the abdomen the right costal groove is found to be partly obliterated. A distinct prominence occupies the right hypochondriac and right half of the epigastric region and extends into the upper half of the umbilical region. Its lower margin, which descends on inspiration, is felt in the median line at the level of the umbilicus; in the mammary line, 10.5 cm. below the costal margin. Its right border cannot be sharply defined. In the median line the fingers can be pressed in above it. Over the resistant mass there is dull tympany, continuous with the hepatic flatness. Leucocytes, 18,300. Urine, clear; specific gravity, 1017; acid. There is no reduction of Fehling's solution. A trace of albumin is present.

On the second day after admission a stool passed was of golden-yellow color. On the third day the leucocytes numbered 19,500, and the temperature varied from 99.2° to 101.8° F. During the night the patient was irrational at times. The temperature rose gradually, reaching a maximum of 104° F. A liquid stool of ochre-yellow color was passed. Urine: specific gravity, 1020; no reaction for sugar was obtained.

The diagnosis of suppurative pancreatitis was made by Dr. Bloodgood, and an operation for its relief was performed under cocaine anæsthesia. A linear longitudinal incision was made below the costal margin within the right mammillary line. After incising the great omentum between the stomach and transverse colon an abscess cavity was entered. Grumous, purulent fluid containing necrotic particles was evacuated. A rubber drainage-tube, packed about with gauze, was inserted into the wound. After operation the pulse remained weak, and death followed at the end of about four hours. The duration of the fatal illness was twenty-one days.

Autopsy. Performed three hours after death. The body is that of a large-framed, muscular man with abundant subcutaneous fat. *Peritoneal cavity:* The omentum, which contains a large quantity of fat, is adherent in the neighborhood of the operation wound to the anterior abdominal wall by light, fibrinous adhesions. Its surface is thickly studded with conspicuous opaque, white areas, varying in size and shape, but usually round and about 3 mm. in diameter, extending about 1.5 mm. below the surface. On section similar foci are found embedded in the fat. Opaque, white areas are present in the fat of the mesentery, where they are most abundant near its intestinal margin, in the subperitoneal fat of the anterior abdominal wall, over the bladder, over the kidneys, and about the colon. *Abscess cavity:* The drainage-tube inserted into the abdominal wound passes through a small, incised opening in the great omentum and enters an immense abscess cavity which occupies approximately the site of the lesser peritoneal cavity. The foramen of Winslow is closed. The stomach is pushed upward and forward, and its anterior surface is adherent to the lower surface of the liver to the left of the gall-bladder. The spleen, partially surrounded by fat containing many opaque, white areas, is bound by fibrous adhesions to the diaphragm. The walls of the cavity are very irregular and ragged, and have a necrotic appearance, in general opaque and gray, occasionally black. This blackish-gray appearance extends only a short distance below the surface, and where the wall is formed by fat give place to numerous foci of opaque, white color. The retroperitoneal fat in front of the left kidney and psoas muscle has been eroded, and an extension of the cavity passes behind the jejunum near its junction with the duodenum. To the left of the descending portion of the duodenum, occupying the position of the pancreas and projecting forward into the abscess cavity, is a great mass of black material, necrotic in appearance, extending to the left as far as the spleen. This material is reddish-black on section, somewhat spongy in texture, soft, dry, and friable. The cavity contains a large quantity—at least 500 c.c. of fluid reddish-gray material, containing fat droplets and black, necrotic particles.

Liver. The organ is flaccid in consistence; the cut surface has a brownish-red color; lobulation is well marked. The bile-ducts are slightly dilated, and contain thin, yellow bile. The gall-bladder is

bound by numerous adhesions to the duodenum and stomach. Its walls are thickened, and it is much distended, containing viscid, yellow bile and a large number—over one hundred—of brown, faceted calculi, varying in diameter from 0.5 to 1 cm. The hepatic, cystic, and common ducts are much dilated. On opening the duodenum a stone is felt below the mucous membrane, situated in the common bile-duct, about 1.5 cm. from the orifice of the diverticulum of Vater. It is 7 mm. in diameter and resembles those present in the gall-bladder. *Pancreas:* The pancreatic duct unites with the common bile-duct at a point 7 mm. from the duodenal orifice. For a distance of 1.5 cm. the two ducts are separated by a thin septum only, and in contact with this septum is lodged the gallstone previously mentioned. The pancreatic duct is not distended. The pancreas occupies the posterior wall of the lesser peritoneal abscess cavity, and is covered by the mass of reddish-black, friable material, changed coagulated blood, above described. The organ is of large size, and the glandular tissue is in great part firm, yellowish-white, and well preserved. The interstitial tissue has a dull reddish, in places hemorrhagic, appearance, and contains conspicuous opaque, yellow areas of irregular shape. Where the anterior surface of the head and body is in contact with the overlying material there is a superficial zone of soft, grayish, necrotic appearance.

The other organs—heart, lungs, spleen, stomach, intestines, and kidneys—present no noteworthy alteration.

Histological Examination. The foci of fat necrosis consist of a central mass of necrotic material, in which the outlines of the fat cells are in most places preserved, though the nuclei have disappeared, and a peripheral zone of cell proliferation. The latter consists in part of large, round cells, with round or slightly irregular, deeply staining nucleus and granular or vacuolated cell body. Multinuclear cells of similar appearance are present. About neighboring bloodvessels lymphoid and plasma cells are accumulated in moderate number, together with an occasional polynuclear leucocyte.

The interstitial tissue of the pancreas is much increased, wide bands of fibrous tissue containing in places lymphoid and plasma cells, separate groups of lobules. Numerous irregularly shaped cells filled with brownish-yellow pigment granules, which give the Prussian-blue reaction for iron, afford evidence of former hemorrhage. In a few places well-preserved red corpuscles are diffusely scattered in the tissue. Foci of necrotic fat are present. Many acini are widely dilated; their cells are flat and the lumen is much distended, containing products of secretion and occasionally one or more polynuclear leucocytes. In an area corresponding to the zone of necrotic appearance before mentioned, nuclei no longer stain, and the architecture of the glandular tissue is only obscurely distinguishable. A thick band of newly formed fibrous tissue containing an occasional acinus or duct separates the necrotic parenchyma from that which is still intact. The mass covering the pancreas is found to consist of altered blood; upon and immediately below its surface are numerous polynuclear leucocytes.

Sections of the diaphragm, stomach, and liver where they form part of the abscess wall show a zone of newly formed fibrous tissue, upon the surface of which are necrotic material and polynuclear leucocytes in great number.

Bacteriological Examination. Cultures from the blood contained in

the heart, from the lung, and from the liver, studied by Mr. V. H. Bassett, were found to contain the bacillus coli communis. A plate culture from the material covering the pancreas, and forming part of the abscess wall, contained the B. coli communis, the B. lactis aërogenes, and the B. proteus vulgaris.

Anatomical Diagnosis. Cholelithiasis; calculus lodged in the common duct near its orifice; slight jaundice. Old hemorrhage within and about the pancreas; localized necrosis of pancreas; chronic interstitial pancreatitis; necrosis of fat of pancreas, greater and lesser omentum, mesentery, and subperitoneal tissue of abdominal wall. Peripancreatic abscess limited by lesser peritoneal cavity. Laparotomy wound.

Symptoms occurring six months before the fatal illness indicated the passage of a calculus along the common duct, and this diagnosis was confirmed at autopsy by the presence of numerous calculi in the gall-bladder. The second attack began suddenly, three weeks before death, with severe nausea and vomiting, accompanied by intense, cramp-like pain in the abdomen. Tenderness was present in the right hypochondriac region, and on admission to the hospital the patient was slightly jaundiced. A mass was felt in the epigastrium; there was irregular fever and a leucocytosis of 18,000 to 19,000. The diagnosis of acute suppurative pancreatitis was made.

The autopsy disclosed a small gallstone lodged in the common bile-duct, a short distance from its orifice, in such a position that it might readily have compressed and occluded the duct of Wirsung, here separated from the bile-duct by a thin, almost membranous septum. The lesser peritoneal cavity was the site of a large abscess, whose walls were formed in large part by necrotic fat; the subperitoneal tissue was studded with conspicuous opaque, white foci of fat necrosis. The pancreas was in great part well preserved, but its interstitial tissue was increased and gave evidence of having been the seat of hemorrhage. The anterior surface of the pancreas was covered by a considerable quantity of old, clotted blood, having the dark appearance of gangrenous tissue and forming the tumor mass, which was palpable during life. A superficial zone of gland parenchyma in contact with this material was necrotic. There was evidence of duct obstruction; though the larger ducts were not dilated, many alveoli were dilated, and there was an increase of interstitial tissue.

Since jaundice was slight and the pancreatic duct was not dilated, it is probable that the calculus produced only temporary obstruction of the two ducts. Nevertheless, one may fairly assume that the primary cause of the lesions in and about the pancreas was an interference with the outflow of the pancreatic secretion. In support of this conclusion I have been able to collect from the literature a number of cases in which, associated with a lesion of the pancreas or with necrosis of the

surrounding fat, usually with both conditions, a gallstone was found lodged in the common duct near its orifice, where it could compress the pancreatic duct, or, perhaps, free in the duodenum, the common duct being dilated (Cases I. and V.). In the latter case there could be little doubt that it has shortly before occupied the first-named position.

Case I.[1]—Male, aged sixty years. During sixteen months the individual had suffered attacks of pain, followed by jaundice. He was suddenly attacked with severe pain in the left hypochondriac and epigastric regions. Collapse preceded death, which followed within thirty-four hours after the onset of symptoms. The thickened gall-bladder contained over a hundred calculi. The common bile-duct was dilated to the size of the little finger, and in the duodenum was a calculus the size of a hazel-nut. The pancreas was large, grayish-pink or reddish-gray, mottled with reddish-brown. Surrounding fat was moderately infiltrated with blood, and contained foci of necrosis.

Case II.[2]—Male, aged forty-nine years. For several months the patient had suffered with pain in the epigastrium, at times in the right hypochondrium. His stools were occasionally clay-colored, and once he was jaundiced. About sixteen hours before death he was suddenly attacked with vomiting and severe epigastric pain, followed by collapse. "The gall-bladder contained one small concretion of inspissated bile; two others were lodged in the duodenal extremity of the common duct." The body and tail of the pancreas were enlarged and infiltrated with blood, exuding a chocolate-colored fluid. In the neighborhood of the gland were foci of fat necrosis.

Case III.[3]—No clinical history is given. In the common duct close to its orifice was a gallstone; others are present in the duct behind it and in the gall-bladder. The body and tail of the pancreas were beset with green and yellow points, the tail being almost completely necrotic. Old and recent hemorrhages occur. Microscopical examination demonstrated the presence of suppurative inflammation, having its origin apparently in the ducts.

Case IV.[4]—Female, aged fifty-eight years. For fifteen years she had suffered with severe attacks of indigestion, on one occasion followed by jaundice; pain in the right hypochondriac region occurred during the later attacks. Four days before death she was seized with pain in the right hypochondrium, accompanied by vomiting, chill, and fever, and followed by jaundice. A small gallstone was found at the outlet of the common duct; others were present in the gall-bladder. The pancreas was enlarged, surrounded and infiltrated with blood. Numerous foci of fat necrosis were seen in its neighborhood.

Case V.[5]—Female, aged forty-eight years. The patient stated that she had suffered for years with stomach and liver trouble. Illness began five days before death, with vomiting, constipation, and abdominal distention; jaundice followed. The thickened gall-bladder contained three large concretions. In the duodenum was a stone the size of a

[1] Thayer. Boston Med. and Surg. Journ., 1889, vol. cxxi. p. 506.
[2] Day. Ibid., 1892, vol. cxxvii. p. 569.
[3] Dieckhoff. Festschr. f. Prof. Thierfelder. Leipzig, 1895.
[4] Cutler. Boston Med. and Surg. Journ., 1895, vol. cxxxii. p. 354.
[5] Fraenkel. Münchener med. Wochensch., 1896, vol. xliii. pp. 814, 844.

cherry, and the papilla biliaria was gaping. In the interstitial tissue of the pancreas, which was fairly firm, were foci of fat necrosis; otherwise the organ appeared to be normal. Areas of necrosis were present in the fat of the greater and lesser omentum and of the appendices exploica.

CASE VI.[1]—Female, aged thirty-eight years. The individual was suddenly seized forty-two hours before death with vomiting and pain in the upper part of the abdomen, followed by symptoms of collapse. A gallstone, about the size of a pea, was found projecting from the orifice of the common duct into the duodenum. Other calculi were found in the gall-bladder and common bile-duct. The pancreas was enlarged, and its appearance of intense injection was evidently due to inflammatory change. Slight peritonitis was present.

CASE VII.[2]—Male, aged forty-one years. The patient was suddenly attacked with vomiting and severe abdominal pain. Collapse preceded death, which occurred at the end of forty-four hours. A small, cubical gallstone projected from the end of the common duct into the duodenum; similar concretions were found in the gall-bladder. The pancreas was greatly enlarged, weighing 525 grammes, and mottled with areas of reddish-brown color, due to hemorrhagic infiltration of its interstitial tissue. The organ was surrounded by semifluid, clotted blood, of dark-brown color. The intima of the splenic artery was of a dirty-blue color, and apparently necrotic about 2 cm. from the aorta; the writer regards this vessel as the source of the hemorrhage, although it is not stated that rupture had occurred.

In the preceding eight cases a calculus had lodged in the common bile-duct near its orifice, or, having been expelled from this position shortly before death, lay in the duodenum, leaving the common duct dilated. Necrosis of the fat in or about the pancreas occurred in seven of these eight cases; in the report of the eighth case (No. 6), briefly described several years after its occurrence, no reference is made to this change. Even in the absence of gross lesions of the gland, as in Case No. 5, the occurrence of fat necrosis is not surprising, since the experiments previously mentioned have shown that simple duct ligation is followed by this change. In the remaining cases macroscopical alterations were observable.

In five instances recent hemorrhages had occurred into the organ, but, microscopical examination being omitted, it is difficult to determine whether these cases should be classified as examples of simple hemorrhage or of hemorrhagic pancreatitis. In the case which I have recorded there was evidence of past hemorrhage into and about the pancreas, accompanied by necrosis of a small portion of the organ. The remainder of the gland was the seat of chronic interstitial change, and apparently, as the result of invasion of micro-organisms into the hemorrhagic and necrotic tissue, an abscess, limited by the lesser peri-

[1] Kennan. Brit. Med. Journ., 1896, vol ii. p. 1442.
[2] Simpson. Edinburgh Med. Journ., 1897, vol. ii. p. 245.

toneal cavity, had formed about the organ. In Case No. 3 there were old and recent hemorrhages into the pancreas, which had undergone suppurative inflammation and necrosis.

Of special interest are the four cases (Nos. 1, 2, 6, and 7) in which death occurred within thirty-three, sixteen, forty-two, and forty-four hours after the onset of sudden vomiting, pain in the upper abdomen, and collapse. The brief duration of symptoms indicates that the calculus found at autopsy had been lodged a short time only in the common duct. Nevertheless, extensive lesion of the pancreas, hemorrhagic in character, had resulted. In the study of reported cases it is often difficult to distinguish between hemorrhage into the pancreas and hemorrhagic pancreatitis. Indeed, the relation which the two conditions bear to one another is not clear. Various writers discuss the possibility of hemorrhage without pre-existing inflammation. Is hemorrhage the result of preceding inflammatory changes, or is inflammation a consequence of the secondary infection of an organ already the seat of hemorrhage? Histological and experimental studies have failed to explain the etiology of the two conditions.

Ligation of the pancreatic ducts in animals does not cause hemorrhage or hemorrhagic inflammation. By injecting the bacillus pyocyaneus and the bacillus diphtheriæ into the pancreatic duct, Flexner[1] obtained intensely hemorrhagic inflammation of the gland in several instances within forty-eight hours. It is conceivable that partial occlusion of the pancreatic duct associated with the invasion of the retained secretion by micro-organisms from the duodenum might cause a similar hemorrhagic lesion.

Should a calculus lodge so firmly in the common bile-duct as to cause obstruction to the outflow of the pancreatic secretion, one would also expect occlusion of the bile-duct and consequent jaundice. Where death rapidly follows, as in Cases Nos. 1, 2, 6, and 7, its absence is not surprising. Jaundice was present in Cases Nos. 4 and 5; in the case which I have recorded it was present in slight degree. The occurrence of advanced lesions in cases which have died within forty-eight hours after the onset of symptoms shows that a profound alteration might be produced by the transient impaction of a calculus, perhaps of insufficient duration to produce jaundice.

The preceding cases include only those in which there is positive evidence that a stone passing along the common bile-duct had become impacted in such position that occlusion of the pancreatic duct might ensue. A calculus might be temporarily so located and cause severe injury to the gland, but finally, being expelled, give no evidence of its former impaction. I have collected from the literature a number of cases in which pancreatic lesions and fat necrosis were associated with

[1] Contributions to the Science of Medicine. Dedicated to Wm. H. Welch, M.D., Baltimore, 1900, p. 743.

cholelithiasis. Gallstones were found in the gall-bladder or gall-ducts, but at autopsy there was not convincing evidence, as in the previously cited cases, that a stone in the common duct had occluded the pancreatic duct. In one of these cases (No. 9) a calculus had found its way into the pancreatic duct and caused suppurative inflammation.

No.	Author.	Duration.	Pancreas.	Fat necrosis.
1	Thayer[1]	34 hours	Hemorrhagic	Present.
2	Day[2]	16 hours	Hemorrhagic	"
3	Dieckhoff[3]	.....	Hemorrhagic, purulent, and gangrenous	"
4	Cutler[4]	4 days	Hemorrhagic	"
5	Fraenkel[5]	5 days	.....	"
6	Kennan[6]	42 hours	Hemorrhagic.	
7	Simpson[7]	44 hours	Hemorrhagic	Present.
8	Opie[8]	21 days	Hemorrhagic and gangrenous	"
9	Dieckhoff[3]	2 days	Purulent	"
10	Rolleston[9]	2 days	Hemorrhagic	"
11	Paul[10]	30 hours	Hemorrhagic.	
12	J. A. Smith[11]	24 hours	Hemorrhagic.	
13	Chiari[12]	7 days	Gangrenous	Present.
14	Chiari[13]	3 weeks	Gangrenous	"
15	Mader[14]	13 days	Gangrenous	"
16	Fitz[15]	10 days	Gangrenous	"
17	Simon and Stanley[16]	3 days	Purulent	"
18	Morian[17]	27 days	Hemorrhagic and gangrenous	"
19	Habersohn[18]	4 months	Gangrenous.	
20	Chiari[13]	2 months	Gangrenous.	
21	König[19]	32 days	Gangrenous	Present.
22	Rolleston[9]	2½ months.	Purulent	"
23	Hawkins[20]	4 days	.....	"
24	Körte[21]	4 months	Gangrenous.	
25	Körte[21]	1 month	Hemorrhagic and gangrenous	Present.
26	Stockton & Williams[22]	3 weeks	.....	"
27	Fraenkel[23]	.....	Gangrenous	"
28	Fraenkel[23]	5 days	Hemorrhagic and gangrenous	"
29	Flexner[24]	.....	.....	"
30	Ehrich[25]	1 month	Gangrenous	"
31	Scott[26]	15 days	Gangrenous	"
32	Grawitz[27]	32 days	Gangrenous	"

[1] Boston Medical and Surgical Journal, 1889, vol. cxxi. p. 506.
[2] Ibid., 1892, vol. cxxvii. p. 569.
[3] Festschrift f. Prof. Thierfelder, Leipzig, 1895.
[4] Boston Medical and Surgical Journal, 1895, vol. cxxxii. p. 354.
[5] Münch. med. Wochensch., 1896, vol. xliii. p. 813.
[6] British Medical Journal, 1896, vol. ii. p. 1442.
[7] Edinburgh Medical Journal, 1897, vol. ii. p. 245.
[8] See above.
[9] Transactions Pathological Society, London, 1893, vol. xliv. p. 71.
[10] Boston Medical and Surgical Journal, 1894, vol. cxxx. p. 8.
[11] British Medical Journal, 1897, vol. ii. p. 468.
[12] Wiener med. Wochensch., 1876, vol. xxvi. p. 291.
[13] Ibid., 1880, vol. xxx. pp. 139, 164.
[14] Bericht d. k. k. Krankenanstatt Rudolfs-Stiftung, 1884; 1885, pp. 371, 435.
[15] Medical Record, 1889, vol. xxxv. pp. 197, 225, 253.
[16] Lancet, 1897, vol. i. p. 1325.
[17] Münch. med. Wochensch., 1899, vol. lxvi. p. 348.
[18] Diseases of the Abdomen, 1878, 3d ed., p. 338.
[19] Inaug. Diss., Kiel, 1889.
[20] Transactions Pathological Society, London, 1893, vol. xliv. p. 78.
[21] Arch. f. klin. Chir., 1894, vol. xlviii. p. 721.
[22] American Journal of the Medical Sciences, 1895, n. s., vol. cx. p. 251.
[23] Münch. med. Wochensch., 1896, vol. xliii. pp. 813, 844.
[24] Journal of Experimental Medicine, 1897, vol. ii. p. 413.
[25] Beiträge z. klin. Chir., 1898, vol. xx. p. 316.
[26] American Journal of the Medical Sciences, 1899, n. s., vol. cxviii. p. 414.
[27] Münch. med. Wochensch., 1899, xlvii. p. 813.

The first eight cases of the table are those already cited.

A hemorrhagic lesion of the pancreas, hemorrhage or hemorrhagic pancreatitis, was present in Cases Nos. 10, 11, and 12. Gallstones were found in the gall-bladder or in the bile passages, but there is no record that a calculus was located near the orifice of the common duct. Jaundice was absent, and the autopsy gave no positive evidence that a stone had recently passed into the duodenum. Death followed within forty-eight hours the onset of symptoms, abdominal pain, vomiting, and collapse. The clinical and pathological picture agrees with that of the preceding cases of rapidly fatal hemorrhagic lesion (Nos. 1, 2, 6, and 7). One factor alone is absent: the offending calculus has apparently been passed into the intestine and lost.

When, it may be, with a history of previous gallstone colic, the fatal attack is accompanied by evidence of the passage of a calculus along the common duct, notably pain and jaundice, and calculi are found in the bile passages after death, though none are found near the orifice of the common duct, one is justified, in view of the more conclusive cases mentioned (Nos. 1 to 8), in assuming that the pancreatic lesion was caused by a calculus temporarily occluding both ducts, but finally expelled and evacuated. The reports of cases Nos. 13 to 18 show that they fulfil these conditions; in all of them well-marked jaundice was present.

In the remaining cases (Nos. 19 to 32), although death did not follow shortly after the onset of symptoms, jaundice was not present, and the clinical history gives insufficient evidence that a stone had passed along the common duct. It is not improbable that in some of these cases the association of pancreatic lesion and cholelithiasis was accidental. On the other hand, it is well known that jaundice does not always follow the passage of a calculus through the common duct. Cases cited above have shown that extensive hemorrhagic lesion of the pancreas may be caused by a stone which has only a short time occluded the pancreatic duct. Where death has not followed rapidly the onset of symptoms, gangrenous pancreatitis, often with evidence of old hemorrhage, is observed.

The cases of acute pancreatic lesion recorded in the literature are not numerous; the relatively large number which have been found associated with cholelithiasis indicates an intimate relation between the two processes, and in view of the facts above noted it is fair to assume that with few exceptions the pancreatic lesion in the collected cases was caused by the presence of gallstones. They number thirty-two.

In twenty-six cases fat necrosis was present. In three of the six cases where it was absent (Nos. 6, 11, and 12) death occurred very soon after the onset of symptoms. In four instances (Nos. 5, 23, 26, and 29) fat necrosis was present, though no gross lesion of the pancreas was noted.

In seven cases (Nos. 1, 2, 6, 7, 10, 11, and 12) death followed the onset of symptoms—abdominal pain, vomiting, and collapse—within forty-eight hours, and at autopsy hemorrhagic infiltration of the pancreas was present. In an eighth case (No 4) death followed in four days. In these cases, as mentioned above, it is not possible to determine whether the gland was the seat of simple hemorrhage or of hemorrhagic pancreatitis. It is probable that the lesion is that usually described as hemorrhagic pancreatitis; here the organ is infiltrated with blood, and there is necrosis of greater or less extent combined with inflammatory changes.

In the great majority of the cases where the fatal illness was of longer duration the pancreas presented the lesion usually described as gangrenous pancreatitis. This condition was present in seventeen cases. The organ is dry, friable, blackish, and necrotic in appearance. Old, changed blood frequently gives evidence of former hemorrhage, and there can be little doubt that this so-called gangrenous pancreatitis represents a late stage of the hemorrhagic lesion. In a large proportion of such cases infection of the injured organ and of the necrotic fat occurs, presumably from the duodenum, and a peripancreatic abscess results, usually limited by the wall of the lesser peritoneal cavity whose fat has become in large part necrotic.

In three cases (Nos. 9, 17, and 22) the pancreas was the seat of suppurative inflammation, apparently without hemorrhage or necrosis. In one of them (No. 9) a gallstone had found its way into the pancreatic duct.

The cases previously cited include only acute lesions of the pancreas. Accompanying long-continued occlusion of the pancreatic ducts one usually observes chronic changes in the gland, chronic interstitial inflammation, dilatation of the ducts, and occasionally the formation of retention cysts. Phulpin[1] has recorded a case in which a stone lodged for a long time near the junction of the common bile-duct and duct of Wirsung occluded both:

A woman, aged seventy-four years, had suffered with jaundice for a year and a half. At autopsy the gall-bladder was found to contain numerous faceted calculi the size of pease. The common bile-duct was so dilated that its diameter almost equalled that of the small intestine. At a point about 2 cm. from its orifice it assumed its usual size, and here was lodged a calculus similar to those found in the gall-gladder. Irregularly scattered in the body and tail of the pancreas were a score of cysts the size of hazel-nuts. The duct of Wirsung was dilated to the size of the index finger. Passing toward the duodenum the dilatation ceased at a point where the pancreatic duct comes in contact with the common bile-duct before traversing with it the wall of the duodenum and opposite the calculus lodged in the common duct. No accessory pancreatic duct could be found.

[1] Phulpin. Bull. Soc. Anat. d. Paris, 1892, vol. lxvii. 5me. S. 6.

In two cases recently reported by Mayo Robson,[1] where operation was performed for removal of gallstones from the common duct, the head of the pancreas was found so indurated that a diagnosis of malignant growth was made. In one of these cases autopsy demonstrated the presence of chronic interstitial pancreatitis. Since in the second case the subsequent history disproved the diagnosis of malignant disease, Robson thinks that here also chronic pancreatitis was present.

In the following case chronic interstitial pancreatitis and fat necrosis was associated with cholelithiasis:

Anatomical Diagnosis. A. G., female, aged fifty years. Cholelithiasis; calculi in gall-bladder, cystic, hepatic, and common bile-ducts. Focal necrosis of liver and acute suppurative inflammation of bile-ducts. Adenocarcinoma of the gall-bladder; metastases in liver. Jaundice. Chronic interstitial pancreatitis. Peripancreatic fat necrosis. Hemorrhage into the gastro-intestinal canal; submucous ecchymosis. Acute spleen tumor; ascites.

Synopsis of Autopsy Protocol. The body is that of a large, well-nourished woman, deeply jaundiced.

The liver, weighing 2250 grammes, has a dark, greenish-yellow color; the bile-ducts are dilated. On the upper surface of the left lobe are several bleb-like projections, the largest 8 mm. across, containing yellow, pus-like fluid. The gall-bladder notch is deep, and the surface of the liver in its neighborhood is much puckered. On freeing adhesions with the duodenum the gall-bladder is found to be firmly contracted over a number of faceted concretions of an average diameter of about 12 mm. The inner surface of the cavity is rough and irregular in shape; its wall where it comes in contact with the under surface of the liver has an average thickness of 6 mm., and is formed by dense, gray-white tissue, which sends short projections upward into the liver substance. Sparsely scattered throughout the liver are small nodules, usually about 5 mm. in diameter, composed of similar tissue. The cavity of the gall-bladder communicates with a second cavity of considerable size, representing apparently the junction of the hepatic, cystic, and common bile-ducts, and containing two faceted stones, each about 1 cm. in diameter. Otherwise the common duct is not markedly dilated, having a circumference of 4 mm., but its walls are somewhat thickened and fibrous.

The pancreas is very firm in consistence, and is infiltrated with and embedded in fat. In the body of the organ are several conspicuous areas of irregular shape, 2 or 3 mm. across, composed of opaque, yellowish-white material. Similar foci of smaller size are sparsely scattered in the interstitial fat throughout the organ. The lumen of the duct of Wirsung is conspicuous, but not markedly dilated.

Histological Examination. Foci of necrosis and of acute inflammation are present throughout the liver. The tissue replacing the gall-bladder where it comes in contact with the liver and the nodules throughout the organ have the structure of adenocarcinoma.

The interstitial tissue of the pancreas is increased throughout the gland, most markedly in the tail, and the normal lobulation of the

[1] Lancet, 1900, vol. ii. p. 235.

gland is much more distinct than usual. Fat is very abundant, and contains foci of necrosis surrounded by cells of lymphoid and epithelioid type in moderate number, together with many polynuclear leucocytes. The medium-sized ducts are much dilated, their cells are flattened, and they contain products of secretion, desquamated cells, and occasionally a few polynuclear leucocytes. In general the acini are normal in appearance, but many are considerably dilated and contain an occasional polynuclear leucocyte.

In the preceding case the changes in the pancreas are those which usually follow duct obstruction; the ducts throughout the gland are dilated, the interstitial tissue between groups of lobules is much increased, and contains foci of fat necrosis. The common bile-duct, which is the seat of chronic inflammation, contains numerous calculi, and one may readily believe that the pancreas has suffered during the expulsion of gallstones.

The preceding study has shown that where anatomical conditions are favorable disease of the pancreas may occur as a complication of cholelithiasis when a calculus passes along the common bile-duct. The lodgement of a stone near the orifice of the bile-duct, where it may at the same time compress and occlude the duct of Wirsung, is not uncommonly a cause of pancreatic lesions and disseminated fat necrosis. Should a calculus become impacted in this position the preceding cases have shown that one of several conditions may result:

1. An individual, usually in fairly good health, with perhaps a history of previous gallstone colic, is suddenly attacked with pain in the epigastric region, accompanied by vomiting and followed by collapse. Death follows usually within forty-eight hours, and at autopsy gallstones are found in the bile passages, while that one which caused the fatal attack may be still lodged in the common duct near its orifice. The pancreas is enlarged, infiltrated with blood, and hemorrhage may have occurred into the surrounding tissue. Foci of fat necrosis are usually present.

2. A fatal termination may not follow rapidly the symptoms mentioned. Pain in the epigastrium persists, jaundice may be present, and a tumor mass above the umbilicus may indicate a probable lesion of the pancreas. At the end of one or more weeks or months death occurs, often with symptoms indicating the presence of suppurative inflammation, presumably in the neighborhood of the gland. At autopsy the diagnosis of cholelithiasis is confirmed by the presence of gallstones in the gall-bladder or in the bile-ducts, and occasionally the offending calculus is still lodged near the junction of the common bile-duct and the duct of Wirsung. The pancreas is dry, black, and necrotic, and evidence of previous hemorrhage may be present. Secondary infection has occurred and the pancreas lies in an abscess cavity formed by the bursa omentalis. In the wall, and often widely disseminated in the

abdominal fat, are foci of necrosis. Since the individual has survived the primary lesion opportunity has been given for the development of secondary changes in the injured pancreas and neighboring fat.

3. In certain instances long-continued or repeated obstruction of the pancreatic duct by gallstones does not cause the acute lesions described, but produces chronic inflammatory changes.

A REPORT OF A CASE OF TYPHOID PLEURISY.

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Pleurisy is not a common complication of typhoid fever; when present it is ordinarily secondary to pneumonia, to an infarction, or to gangrene. Primary pleurisy is regarded as very rare. Bacteriological study usually shows the presence of the more common members of the pyogenic group of micro-organisms. However, a few isolated reports in literature demonstrate the etiological relationship of the typhoid bacillus to certain cases. Thus fifteen years ago Rendu and de Gennes[1] claimed to have isolated the bacillus typhosus from the pus of an empyema. Two years later A. Fraenkel[2] reported a similar case. These reports were followed by others. In 1889, Valentini[3] isolated the typhoid organism from a purulent pleural exudate; then Mya and Belfanti,[4] in 1890, from a pleural abscess; and Weintraud[5] still later from a case of purulent pleurisy. More recently other examples have been published by Dineur,[6] Gerhardt,[7] and Souques, Lesnè and Ravaut.[8]

The history of the case reported by us is as follows:

The patient, a physician, aged fifty-seven years, was seen by Dr. Gordinier in consultation at Schaghticoke, N. Y., July 14, 1899. He presented the following history: In 1858, was ill several weeks with inflammatory rheumatism. In 1862, had, while in Virginia, typhus

[1] Rendu and de Gennes. La France Médicale, 1885, vol. ii. p. 1821.
[2] A Fraenkel. Verhandlung sechste Kongress f. inner. Med., 1887, p. 179.
[3] Valentini. Berl. klin. Wochenschrift, 1889, p. 368.
[4] Mya and Belfanti Giornale d. Acad. di Med. di Turino, January, 1890.
[5] Weintraud. Berlin. klinische Wochenschrift, 1893, p. 345.
[6] Dineur. Bull. de l'Acad. Royale de Med. de Belgique, 1897.
[7] Gerhardt. Mittheilungen a. d. Grenzgebieten der Med. u. Chir., Bd. 1900, Heft 1.
[8] Souques, Lesnè and Ravaut. Bull. et Mém. de la Soc. Med. de Paris, January 25, 1900.

fever. In 1885, was sick four months with what was diagnosed as typhoid fever; since that illness has been in comparatively good health, save that he has suffered frequently with labored breathing on ascending hills or stairs, or after any violent exercise. Has had on several occasions slight œdema of ankles. On July 10, 1899, patient was obliged to take to his bed, having previously suffered for several days with malaise, loss of appetite, disinclination to work, together with headache, pains in muscles and joints, and colicky pains in abdomen. Bowels were constipated.

Present Condition. Patient slightly built, with ill-developed and flabby musculature. Lips, ears, and fingers very cyanotic, face dusky. Respiration 60, pulse 120, small and irregular, arteries not thickened. Temperature 103°. Tongue coated with yellowish fur. No œdema, veins of chest and abdomen not prominent, abdomen distended, no rose spots. Heart's apex diffuse in left fifth interspace just outside the nipple line, distinct epigastric pulsation. At apex a well-marked presystolic thrill was felt, and there was heard a presystolic murmur, followed immediately by a soft systolic murmur, which latter murmur was conducted to the axilla; second sound at base reduplicated. Liver dulness in fourth interspace extending downward one inch below costal border; splenic dulness distinctly increased, extending downward, forward, and inward. Lungs anteriorly hyper-resonant, with prolonged low-pitched expiration; posteriorly at bases slight dulness, with fine, liquid rales; abdomen very tympanitic; slight tenderness in right iliac fossa.

Diagnosis. Probably typhoid fever, with œdema and congestion of the lungs, the result of cardiac failure. Ordered nitroglycerin, digitalis, and strychnine. On returning home examined the urine, which gave a distinct diazo reaction, and in addition contained a trace of albumin with a few hyaline casts. The blood showed a positive Widal reaction, no leucocytosis, no plasmodia.

July 17th. Visited the patient again and found his general condition much improved, his pulse more regular and less rapid, and his cyanosis disappearing. The heart was more steady, and the sudden sharp slap of the first sound following in the wake of the presystolic murmur was very marked. The liquid râles at the base of the left lung had entirely disappeared, and in their stead a distinct vesicular murmur was detected. At the base of the right chest posteriorly flatness existed, together with absence of voice sounds and vesicular murmur, this flatness extending into the axillary region, where opposite the fifth rib a friction sound was audible. Temperature 102.5° F., and he had had fever continuously since my first visit.

19th. Was called again, and found the physical evidences of a large, right-sided pleural effusion. In consideration of the displaced position of the already crippled heart it was deemed wise to remove the exudate at once. Accordingly I aspirated two quarts of a greenish-yellow, opalescent fluid, with much temporary relief.

24th. Owing to recurrence of extreme dyspnœa he was again tapped, a little less than two quarts of a similar colored fluid being removed. This fluid was examined by Miss Beale, a daughter of the physician in attendance, who found a motile organism in it.

On July 30th I had occasion to again aspirate him, removing twenty ounces of fluid, more milky in character and of much greater consist-

ency. This fluid was received in a sterile vessel and examined microscopically. It contained numerous bacilli actively motile and having the morphological appearances of the typhoid or colon bacillus.

Bacteriological Report. The exudate was placed in a sterile vessel and sent to the Bender Hygienic Laboratory. Four agar-agar plates were then immediately made, using 1 c.cm. of the exudate for each plate. At the end of forty-eight hours of incubation at 37° C. a few discrete, gray, pinhead-sized, finely granular colonies were observed in all the plates. The plate in which they were most numerous contained twenty-six colonies; that containing the smallest number showed eleven colonies. For the most part all the colonies resembled each other in appearance. Many cover-slip preparations were made from different colonies and from the various plates; invariably the same rather short, moderately thick bacillus was observed. When stained by Gram's method this organism was decolorized. On media the various cultures from the different plates presented the following general reactions:

Agar-agar: Moderately abundant, moist, grayish-white growth. No discoloration of the medium.

Gelatin stab: Growth along the entire line of inoculation. No liquefaction of the gelatin at the end of seventeen days.

Potato: Moderate, glistening, gray, moist growth along inoculation tract. No odor.

Litmus milk: Slight acid formation at the end of three days. No coagulation of the milk at the end of fourteen days.

Bouillon: Uniformly cloudy.

Dunham: Same appearance as in the bouillon. No indol at the end of fourteen days.

Glucose, saccharose, and lactose fermentation broth: No gas formation at the end of fourteen days.

Hanging-drop preparations showed actively motile bacilli.

The agglutination-test was tried with the serum of three known cases of typhoid fever; in one case the test was positive in a dilution of 1:110.

Bacteriological Diagnosis. Bacillus typhosus in pure culture.

For the most part the cases thus far reported have occurred in men; usually the left side was affected. Few cases have been reported as occurring early in the attack of typhoid fever; the third week of the disease seems to be the favorite time of onset. One of the most striking features of these pleurisies incited by the bacillus typhosus is the almost uniform character of the pleural exudate. Although hemorrhagic[1] and serofibrinous exudates[2] occur, in the majority of instances the aspirated fluid has been found to be purulent in character. Our own case was no exception to this rule.

[1] Souques, Lesnè and Ravaut. Loc. cit.

[2] Fernet. Soc. Méd. des hôpitaux de Paris, May 15, 1891. Menètrier. Ibid., December 10 1895.

ASTHENIC BULBAR PALSY.[1]

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We will give no historical account of asthenic bulbar palsy because Dr. Henry Campbell and Mr. Edwin Bramwell have only recently, in the summer number of *Brain*, published a careful summary of reported cases, and have made a thorough critical study of the literature. It will suffice to say that the first case was reported by Wilks in *Guy's Hospital Reports* in 1877. Wilks' patient was a stout, well-looking girl who could scarcely walk. The weakness seemed to be due rather to lethargy than to palsy. She spoke slowly, and had slight nystagmus. A month later many of the symptoms of bulbar palsy appeared, coming on rapidly in about three days. She spoke indistinctly, swallowed with difficulty, and was unable to cough. The limbs were not palsied. Soon breathing became difficult, and in a few hours she died. In the then state of knowledge it would have been proper to have expected to find organic bulbar disease. None was present. Careful macroscopical and microscopical examination revealed nothing abnormal. In 1879 Erb published a similar case, and from that time till 1886 nothing more was written upon the subject. About sixty cases have been reported, with twenty-three deaths and seventeen necropsies. These figures give a false impression of the death-rate, and the high percentage is due to the fact that neurologists are prone to report fatal cases, and many non-fatal cases are probably not diagnosed. At first it was thought the disease affected only muscles innervated from the bulb, but in recent years it has been found to be more wide-spread in bodily distribution, and now it is usually described under the title of myasthenia gravis.

For our own work we propose to relate a case that came to necropsy, to discuss another which, though resembling the first in many ways, yet differs enough to make its proper classification doubtful, and to speculate a little, for the matter is still speculative, as to causation.

We are indebted to Dr. Morris J. Lewis for the early history of the first case.

[1] Read at the meeting of the College of Physicians of Philadelphia, November 7, 1900.

A woman, aged nineteen years, and married, came to the dispensary of the Infirmary for Nervous Diseases on April 26, 1899. Her family history was negative, her personal history unimportant, except that ever since her marriage, in 1897, she had had much trouble and grief —an unwished-for pregnancy and a brutal husband. In January, 1898, an abortion, probably induced, occurred. She felt no immediate serious ill effects, but about a year later began to suffer from numbness in the right hand, and later in the left hand, legs, and back. She could feel touch, pin-pricks, and the contact of objects without difficulty. The numbness was entirely subjective. Her condition remained the same until a few weeks before she came to the dispensary. She then rapidly grew weak in the arms and legs, so that she could not easily go up or down stairs, and sometimes fell when walking on a level surface.

Examination. She walked a little stiffly and as well with her eyes closed as open. Station was good. The pupils responded to light. The arms and legs were weak. The dynamometer registered fifty in either hand. The knee-jerks were exaggerated, quick, and equal. There was no ankle clonus. The elbow-jerk was exaggerated. She said she could not feel a sharp prick on the hands or legs, and only slightly on the face. She was, however, able to pick up a small piece of paper even when blindfolded. She could distinguish between hot and cold, but said she felt them but slightly. There was a slight tremor of the hands and head. The eyelids trembled and drooped somewhat, covering about half of the pupil. The lids did not come into close apposition on shutting the eyes. She could raise the lids, but not hold them up long. The tongue was tremulous, and when protruded turned toward the left. The respiration rate was twenty-six. The heart was normal. The sphincters were under complete control. There was slight contraction, but no reversal of the color fields. She was easily hypnotized.

She returned to the dispensary a few times, and then was lost sight of until her admission to Dr. Burr's wards at the Philadelphia Hospital on April 23, 1900.

Examination. A young woman of small frame, moderately well nourished, but somewhat pale. The head fell slightly forward upon the chest, and the eyelids drooped, partially covering the pupils. The face lacked expression. She could lift the head, but said it tired her very much to hold it up. She could not close the eyelids tightly, nor open them completely; there was but little power of movement in the frontal muscles, and she could not wrinkle the eyebrows. Her gait resembled that of a person convalescing from some serious acute disease. The movements of the arms were weak. There was no distinct palsy anywhere. Speech was slow and low-voiced—not aphasic, not paralytic, but simply weak. Mastication was slow, and she complained that eating tired her. Her whole manner and bearing showed languor and weariness, but that something more than mere neurasthenia was present was proven by the fact that the amount of weakness varied greatly in different groups of muscles, being most marked in those innervated from the bulb. The knee-jerks were large, but not spastic, and were easily exhausted. Neither ankle nor patella clonus was present. Babinski's reflex was absent; indeed, the plantar reflex was normal. There were slight but quite constant choreiform movements

in the face and arms. There was no muscular atrophy of the lips, face, or tongue, and of course none in the extremities. She had difficulty in swallowing, solids seeming to stick in the throat and liquids making her cough. She still complained of numbness in the extremities, but felt touch, pain, and heat and cold well. Dr. Charles A. Oliver examined her eyes and reported: "Vision in each eye is reduced one-half to one-third, that of the right being slightly improved by pin-hole. Pupils are equal in size. Irides respond to light, accommodation and convergence. Extra-ocular movements are very much impeded, especially in outward direction, and more marked to the left. Palsy of convergence in association with a variety of ataxic movements. The eye-grounds are healthy, the fields of vision somewhat concentrically diminished. The patient is unable to close the lids, the action of the left orbicularis being more marked." Her condition remained the same until May 13th, when the difficulty in swallowing suddenly became very great, the temperature rose to 101° F., dyspnœa appeared, the pulse was rapid and weak, cyanosis and coma developed, and she died the next morning.

The necropsy was made the following day; it revealed nothing. The thoracic and abdominal organs were normal. There was a small calcified focus of tuberculosis at the apex of the left lung, and the spleen was chronically enlarged and its capsule much thickened. The kidneys and adrenals were not diseased in any way. The right ovary was cystic. The thymus gland was enlarged, but there was no other evidence of lymphatic diathesis. The uterus contained a three or four-months' old fœtus. The brain, after hardening in formalin, weighed 1250 grammes. Neither it nor the meninges showed any change on gross examination. The cerebrum was of full size, but the pons and medulla were distinctly smaller than normal. The pons was one-third less in diameter than others which had been hardened in the same way. The spinal cord was unusually broad in the lumbar region, and after removal of the dura showed a median dorsal fissure starting at the third lumbar segment and extending to the fifth. At first sight there appeared to be a distinct bifurcation of the cord, but section revealed a persistence of the posterior median fissure extending down to the commissure. The other regions of the cord were normal.

Microscopical Examination. Serial sections of the cord, medulla, pons, and floor of the third ventricle were stained by the Marchi, Nissl, Weigert, carmine, and nuclear stain methods. In the cord no pathological changes were found. In the medulla there were distinct chromotalytic changes with swelling and displacement of the nuclei in the cells of the upper nucleus of the tenth nerve (nucleus terminis vagi). The other bulbar nuclei, including the twelfth and nucleus ambiguous, were perfectly normal. We expected possibly to find changes in the seventh and third nuclei, but they were likewise normal. Sections of the cortex revealed no changes by any of the above-named methods. Sections from all the cranial nerves were examined. By the Marchi method black dots were seen scattered here and there throughout the twelfth, tenth, eighth, and fifth nerves; but inasmuch as similar appearances are seen in healthy nerves, or at least in nerves which have performed their functions well, we attribute no significance to them. Examination of the tenth nerve stained with carmine showed an atrophy of some of the nerve fibres, giving the appearance of a sclerosis. In

the tenth and twelfth nerves some swollen axis-cylinders were seen. The muscles showed no pathological changes on microscopical examination.

There are two important questions in the study of this affection: Where is the disease located, and what is its nature? Morbid anatomy gives no help in answering these questions. In the larger number of necropsies no lesion at all has been found, and in the others the changes have been slight and indefinite, similar to those in our case, and of such a nature as not to prove, scarcely to suggest, that they had anything to do with the symptoms. We are compelled, therefore, to fall back upon general physiological and pathological laws, and to draw inferences from analogous affections. The disease must of course be seated in either the muscles or the motor neurons, and if in the latter it must affect either the upper motor neuron from the cerebral cortex to the bulb or cord, or the lower neuron from the bulb and cord to the periphery or some one part, cell body, or axon, of either. The symptoms are so predominately motor that there is no need to look for disease outside the motor apparatus. The disease has been thought to be muscular, but there is much evidence against it. In a large number of cases it has been found that on applying a tetanizing (faradic) current to the muscles at first a brisk contraction is produced, which gradually becomes feeble, and finally ceases, to reappear if the muscle is allowed a period of rest. With the galvanic current, on the contrary, no such exhaustion of muscular contractility occurs. Now faradism causes contraction essentially by acting through the nerves, galvanism by both the nervous system and the muscle directly. Further, Dr. Farquhar Buzzard, at the suggestion of Dr. Campbell, made the following experiment: A moderate galvanic current was applied to the biceps muscle, and a contraction obtained. The muscle was then faradized until it gave no response to a strong stimulus. Then it was tired out by making the patient flex the elbow against resistance, exerted until all power of flexion was lost. On applying the same strength of galvanic current as used at first an excellent contraction was obtained. Finally, on again applying the faradic current, the muscle was found to be still irresponsive. This experiment points strongly against disease of the muscle. Again, we know of no other affection due to disease of the muscles producing analogous symptoms, whereas organic disease of the bulb is not a little similar. Sudden death, a not infrequent thing in asthenic bulbar palsy, is not a symptom of muscular disease. There are several reasons for believing that the upper motor neuron is not affected. The myasthenic reaction, the exhaustibility of the knee-jerk, the pharyngeal palsy, the dyspnœa, the cardiac palpitation, all are evidence against cerebral disease. There is but little direct post-mortem evidence in favor of the bulb as the seat of the disease. The

slight changes that have been found were in it and in the cranial nerves. These changes, though almost certainly insufficient to cause the symptoms, may indicate the action of a toxin. The symptoms certainly point toward the bulb as the part first and most seriously affected. Considering everything we may say with comparative safety that the affection is one of the lower motor neurons, but whether the cell bodies or the axons are first and most affected cannot as yet be determined. The primary seat of disease may be in the motor muscular end-plates.

As to the nature of the disease but little is known. It looks like a toxin disease. It frequently follows some mycotic affection. It kills without visible wound. That poisons arising within or without the body may cause death without producing any discoverable lesion goes without saying. That there are diseases without a visible anatomical basis must be admitted, notwithstanding the dogma that there is never perversion of function without alteration of structure. In our case, as in a few others, we are inclined to believe that pregnancy had some causative influence. She was pregnant at the onset, and again when her disease became manifestly serious. Pregnancy may seriously disturb the normal metabolism of the body, as is shown in the kidney of pregnancy, the multiple neuritis occasionally seen, and the greater predisposition of pregnant women to certain diseases. Again, the smallness of the bulb may have had some influence. This was developmental, not secondary, not due to any acquired disease, not caused by the shrinking of an old sclerosis. What nervous elements were present were normal, but they were fewer than usual, they may have been dynamically weaker. It is possible that the ill-developed bulb was unable to withstand the stress of pregnancy.

The second case is made more difficult to understand by the presence of a singular type of anæsthesia. Sensory symptoms of any kind have been but little pronounced in the cases of asthenic bulbar palsy heretofore observed. Occasionally there is some little aching at the back of the neck and in the shoulders, with, it may be, numbness in the arms. True tactile anæsthesia has never been seen. In the case we are about to relate a very interesting form of anæsthesia was present, namely, asteriognosis, the inability to recognize objects by touch, though simple tactile sense is preserved. This symptom makes the proper classification of the case doubtful; but as it more closely resembles asthenic bulbar palsy than any other disease, we place it tentatively there. It would be interesting if in the future there should be met with cases of anæsthesia in the distribution of the sensory cranial nerves causing a condition comparable or at least analogous to the motor disease, bulbar palsy. This is a speculation, of course, but it is possible that such a condition may sometime be discovered. Our case then would be a connecting link. The history is as follows:

A young woman, aged twenty-four years, well educated and of excellent intelligence, with a good family and personal history, came to Dr. Burr, in March of this year, complaining of general neurasthenic symptoms and marked pseudo-emotionalism. She dated her illness from an attack of grippe which occurred in June, 1899. Whether this attack was one of influenza vera caused by the bacillus of Pfeiffer, or the so-called influenza nostras of unknown causation, could not be determined. At all events she never completely recovered. She was tired, languid, weak, and subject to spells of crying unaccompanied by any distressing emotional feeling. She did not regard her condition as serious, accepted no medical treatment, and grew steadily worse. A week before she came under observation her left arm began to be numb, and she had trouble in picking up small objects. A day or two later the numbness extended to the left leg, and still later to the right side. At the same time her neck felt somewhat stiff and there was severe pain in the occiput. She had slight vertigo, blurred vision, and occasional diplopia.

Examination revealed a spare, rather pale young woman. She was very emotional, bursting into tears without any cause. In the midst of her weeping she would say in the most matter-of-fact way that she did not know what she was crying about, and would go on talking about matters and things in general, paying but little attention to the lachrymal flood. Gait and station were normal; the knee-jerks were normal. Anæsthesia was absent. There was no deformity of the spine, nor pain on pressure anywhere in its length. Her condition was not regarded as serious. A week later, however, she suddenly developed palsy of the right face, involving the entire side. This was not an hysterical spasm, but a true palsy. A few hours later there came on excruciating pain in both ears, which lasted throughout the night, and required morphia for its relief. When re-examined there were found a little drooping of the eyelids, a little trouble in swallowing, slightly nasal voice, weakness of the muscles of mastication on both sides, but the right facial palsy had improved. On rising she would walk well for a few minutes, but soon would stagger and be compelled to sit down. All movements of the left arm were present, but all were weak, and soon she could not move the fingers or wrist against the slightest resistance. She could not squeeze the dynamometer at all. Power in the right arm and hand was a little greater. At no time was there complete loss of power in the arms. She could always make unresisted movements, but could not even lift a spoon, could not feed herself, and even unresisted movements soon tired her so much that she could not continue them. She could not write a word, could not grasp a pen strongly enough to hold it. In bed the movements of the legs were done weakly, but even at the worst she could stand and walk a little. The legs were never so seriously affected as the arms. She could move the tongue in all directions, but complained that the tip felt as if it had been burnt. There was no muscular wasting anywhere. The electrical reactions were normal, but unfortunately no examination was made for faradic exhaustibility. She had frequent attacks of pain in the occiput and back of the left ear, especially if she sat up in bed with the head unsupported. Palpation of the neck and head did not increase the pain at the time, but after an examination pain was apt to occur. No deformity of any kind was discoverable in the occipital region or in the cervical spine. There was no spasm of the muscles of the neck.

The knee-jerks were normal, and ankle clonus was not present. Touch, pain, and temperature-sense were normal on the arms, legs, and face; but later, when the power in the arms had returned and she could grasp well, it was discovered that she could not recognize objects by touch. This test, of course, could not be made while she was unable to handle objects. Smell and taste were normal throughout the course of the disease.

Dr. John T. Carpenter examined her eyes and reported as follows: "Central vision normal; field for form and color strictly normal. No reversal of the fields. No pathological changes in the eye-grounds. Refraction error; moderate hypermetropia. No diplopia except when red glass interposed, when vertical diplopia results and is constant. Right hyperphoria 2°, which is concomitant, not paretic. Ocular movements normal, though both adduction and abduction are below par (15°—5°). The only important factor elicited was the discovery of constant right hyperphoria." The sphincters were under complete control.

On first seeing the patient I did not regard her as seriously ill, but when the facial palsy appeared, followed by the difficulty in swallowing, the nasal speech, and the great muscular weakness, I began to fear a very serious issue, and believed that there was organic disease in the bulb and upper cervical cord or in the neighboring bones. Events proved this was an error, for after a few months she improved rapidly, and now is quite well.

There is no specific in the treatment of asthenic bulbar palsy. Rest is the most important element. The patient should be put to bed; faradism does harm; galvanism is of doubtful benefit; massage is useful. As to medicines, arsenic, in our experience, is the one drug which seems to be of benefit.

A STUDY OF A CASE OF GONORRHŒAL ULCERATIVE ENDOCARDITIS, WITH CULTIVATION OF THE GONOCOCCUS.

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Notwithstanding the wide influence of the original assertion of Bumm that gonorrhœal processes remain limited to mucous membranes, the fact has been clearly established that the gonococcus may, under certain conditions, incite the production of a series of lesions in the body tissues generally, as dissimilar in pathological significance as they are varied in their clinical aspects. Such a relationship has been

well shown for certain gonorrhœal complications of the joints, peritoneum, myocardium, and a few other tissues.

The case herein reported belongs to a small group of cardiac infections induced by the micrococcus gonorrhœæ. Although recognition of the possibility of endocardial complication in gonorrhœa is by no means new, the complete bacteriological proofs of its occurrence are few and recent. The bacteriological study begins with the description of the gonococcus by Neisser in 1879, but more particularly from the cultural studies of Bumm in 1885, and especially those of Wertheim in 1892.

The number of cases in which the proof of the purely gonococcal nature of the endocarditis may really be said to be fully shown is still very small; with my own this does not exceed six cases. Any new contribution of this kind will then have some importance and considerable interest.[1]

For the history of the case I am indebted to the staff of the Roosevelt Hospital. It is as follows:

Lofton J., colored, native of the United States, aged twenty years, single, and a bell-boy by occupation, was admitted to the hospital on August 4, 1900, with loss of speech.

Family History. Unknown.

Personal History. Moderately alcoholic; no history of syphilis; has always been healthy to the time of present illness.

Present Illness. Although no statement was obtained from the patient, it has been ascertained that he had been suffering from urethral disease for the last eight weeks. Six weeks ago he was seized with a chill, followed by fever. Similar recurrences two or three times a week since then. He was forced to give up work, but did not go to bed. He is said to have been irrational on several occasions. This condition continued until yesterday morning, when it was observed that the power of speech was gone.

On entrance (August 4th) the following note was made:

Nutrition good; mucous membrane pale; tongue coated, moist, swollen, and with impressions along the edge; upper lip swollen and tender; pulse rapid, soft, good volume, regular in rhythm; radial artery not thickened; pulse 126; respiration 32; temperature 103° F. Facial expression heavy and dull; no prostration; power of articulate speech gone; occasionally appears to comprehend what is said to him, and will obey. Abdomen and extremities seem normal. On the left side a single inguinal gland is enlarged and firm. Examination of the lungs is negative. The apex beat is in the fifth space in the nipple line. A loud, systolic murmur, which is transmitted to the left, is heard at the apex. The liver does not appear enlarged, nor is the spleen palpable.

[1] Almost the whole bibliography of gonorrhœal endocarditis may be found in Marcel See's Le Gonocoque, Paris, 1895, and Thayer and Lazear's article, A Second Case of Gonorrhœal Septicæmia and Ulcerative Endocarditis, with Observations upon the Cardiac Complications of Gonorrhœa, Journal of Experimental Medicine, 1899, vol. iv. p. 8.

Later. Urine: amber, acid; 1020; no albumin or sugar; urea, 2.1 per cent.; no casts, pus, or blood. Restless, trying to get out of bed; restrained. Involuntary dejection.

August 5th. A purulent urethral discharge was noticed. Same general condition. Iodide of potassium, in 20-grain doses every six hours, and mercury inunctions have been prescribed. (At this time syphilis was suspected.)

6th. Low, muttering delirium continues; takes nourishment badly. Phimosis is present. Circumcision by dorsal incision. Upper lip swollen.

9th. Condition remains unchanged. Urethral irrigations with potassium permanganate instituted.

11th. Patient drank about eight ounces of a 1:40 carbolic solution from an instrument dish. Stomach washed with magnesium sulphate; no harmful effects therefrom.

The daily bedside notes show very much the same condition until August 17th. The urethral discharge ceased and the irrigations were stopped. The temperature-chart up to this time shows an irregular curve, ranging from 99.5° to 103.4° F.

18th. The blood was examined, with the following result: Hæmoglobin, 80 per cent.; red cells, 4,106,000; white cells, 11,400.

24th. Condition the same. This morning the right elbow-joint was noticed swollen, hot, painful, and tender.

25th. The potassium iodide has been increased to 75 grains.

26th. The elbow-joint is less swollen, but still painful.

27th. The elbow swelling has disappeared, but some pain and tenderness still persist.

31st. Blood examination shows: Hæmoglobin, 54 per cent.; red cells, 3,128,000; white cells, 10,800.

Emaciating rapidly. All the way through the pulse has been between 100 and 150, usually above 130.

This clinical picture persisted until September 5th. The temperature, however, which has ranged from 100° to 102° F., went up on September 3d to 103° F., and the following day to 104° F. On the day of his death, which occurred September 5th at 7 P.M., the temperature was 103.8° F. During the day there had been some epistaxis and also bleeding from the mouth. The post-mortem examination was made fifteen hours after death. The following notes have been taken from the autopsy protocol:

Anatomical Diagnosis. Ulcerative endocarditis of the mitral valve, urethritis, acute splenic tumor with infarction, cloudy swelling of the liver and kidneys, œdema, and congestion of the lungs.

Body of a well-built, moderately emaciated negro. No subcutaneous œdema; no apparent enlargement of the superficial glands. Pupils moderately dilated and equal. Mucous membranes pale. Circumcision wound healed; no scars on glans. Subcutaneous fat pale and scanty.

Abdomen. Both layers of peritoneum smooth; no excess of fluid in the peritoneal cavity; foramen of Winslow patent. Appendix vermiformis passes upward behind the cæcum. Omentum dilated; glands not enlarged; right lobe of the liver extends 3 cm. below the costal margin. Diaphragm: fifth rib on the right side, sixth on the left in the mammary lines.

Both pleural cavities free from fluid.

Pericardium. Both layers smooth and normal; cavity contains about 50 c.c. of clear, yellow fluid.

Heart. Distended with fluid blood and chicken-fat and red post-mortem clots. The endocardium and valves on the right side present no abnormality. Adherent to the margin of the posterior segment of the mitral valve is an extensive and irregular but firmly attached thrombus. At the site of attachment the valve is in part destroyed. The chordæ tendinæ of the involved mitral segment are also in part covered with smaller thrombi; the same is also true of the papillary muscle. The measurement of the projecting thrombus on the mitral valve is variable; in some portions this is 1 cm. The average scarcely exceeds 0.5 cm. The myocardium is firm and, on section, of a homogeneous brownish-red color. Eccentric hypertrophy of the left heart. Coronaries normal.

Lungs. Both lungs present a similar appearance. The pleuræ are smooth; the lungs congested and œdematous. Bronchi contains a small amount of frothy fluid. Bloodvessels normal.

Spleen. Enlarged (13 x 8 x 5 x 6 cm.), soft, capsule smooth. On section, chocolate color; Malpighian bodies swollen, pulp increased, trabeculæ apparently not augmented. In the outer half in the upper quadrant of the organ a large yellow wedge-shaped area of tissue replaces that of the spleen proper (infarct).

Liver. Free from adhesions, softer than normal, and on section cloudy. Gall-bladder normal. Common duct patent.

Kidneys. Similar condition on both sides. Fatty capsule scanty, fibrous. Capsule strips off easily. On section, cortex markings indistinct; glomeruli moderately prominent; medulla and pelvis apparently normal. Both ureters, adrenals, testicles, the aorta, pancreas, œsophagus, stomach, intestines, bladder, larynx, and trachea normal.

The floor of the deep spongy urethra presents a small granulating patch covered with a small amount of pus.

Brain normal. Cord not examined.

Microscopical Examination. The histological study of the various tissues confirmed the gross findings. In the alveoli of the lungs a small number of red cells and a few leucocytes were present as well as a moderate number of desquamated epithelial cells and granular material. The microscopical picture of the spleen was typically that of acute splenic tumor; the infarct presented no noteworthy features. Sections of the spleen and portions of the infarct stained for bacteria with Loeffler's methylene-blue and Sterling's gentian-violet failed to show any micro-organisms. In the kidneys two or three areas of round-cell infiltration were seen in the cortex, otherwise the lesion was merely one of albuminous degeneration.

The affected portion of the urethra showed losses of epithelium, with considerable round and polymorphonuclear-cell infiltration in the tissue underneath. A considerable number of micrococci arranged in pairs, with the characteristic morphology of the gonococcus, were present on the surface and in the inflamed tissue. They decolorized by Gram, and some were noted with an intracellular distribution within polymorphonuclear cells. Other cocci, oval in form and not decolorizing by Gram, were likewise present, having an extracellular position.

Heart. The myocardium showed nothing further. Sections through the affected mitral valve and adhering thrombus present several noteworthy features. The thrombus consists of masses of fibrin, some red

cells, and many leucocytes. Portions of the valvular connective tissue show small areas of necrosis along the margin, and considerable numbers of round cells; here the leucocytic infiltration is very great. Sections stained by Weigert's modification of Gram's method show no bacteria. Stained with Sterling's gentian-violet or Loeffler's methylene-blue a few "biscuit-shaped" cocci, for the most part arranged in pairs, may be observed occasionally within the polymorphonuclear leucocytes, more often extracellular.

Bacteriological Report. Tube and plate cultures from the heart's blood, lungs, spleen, liver, splenic infarct, right kidney, gall-bladder, urinary bladder, and bone-marrow were made upon solidified blood-serum (bullock's) and agar-agar.

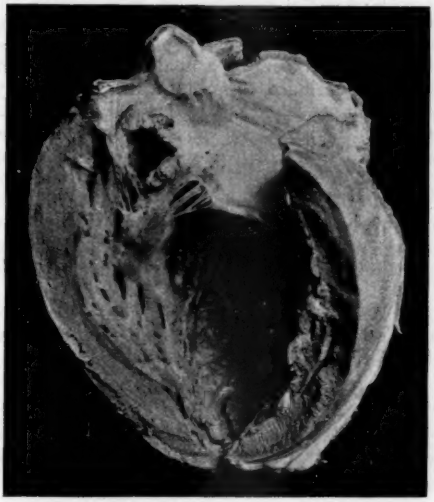
Particular attention was given to the cultivation of micro-organisms from the valvular vegetations. After searing the surface of the latter very thoroughly cultures were made from several places upon agar-agar, solidified blood-serum, Laitinen's medium,[1] and in a mixture of ascitic fluid and ordinary peptone agar-agar containing 2 per cent. glucose. The ascitic fluid and agar-agar were mixed in equal parts. In this series of cultivations from the heart lesion those upon simple agar-agar proved negative. In one blood-serum tube two small gray, discrete, pinpoint-sized colonies were observed at the end of forty-eight hours. Cover-slips showed them to consist of diplococci, often having the characteristic "biscuit" morphology. They decolorized by Gram. Transplantation to ordinary agar-agar gave no results at the end of one week in the incubator at 37° C.; when transplanted upon Laitinen's medium, or to the mixture of sugar agar-agar and ascitic fluid, a scanty growth could be obtained, made up of cocci presenting the same general morphological and tinctorial character. In three of the plates containing Laitinen's mixture, growths consisting of six to eleven discrete white or gray colonies were obtained; the two others were still sterile at the end of nine days. Of the four plates of the sugar agar-agar and ascitic fluid mixture one gave one colony and another three colonies. Both were made up of cocci similar in all respects to those of the other cultures. Subcultures in peptone bouillon, glycerin broth, gelatin, on peptone agar-agar, potato, and glucose agar-agar were invariably negative. (The reaction of these media (potato excepted) was 1.5 + to phenolphthalein.) Transplantations to solidified blood-serum (bullock's) gave variable results. There were as many failures as positive results. When successful their growth was very scanty and the viability short. Cover-slip preparations invariably showed the same moderate sized coccus, generally with diplo arrangement, and often with the "biscuit" morphology. They stained readily with gentian-violet, fuchsin, and Loeffler's methylene-blue; decolorization with Gram was prompt.

Animal Experiments. A suspension of four colonies was made in 5 c.c. of sterilized peptone broth and the whole inoculated intravenously in equally divided doses into two young rabbits (787 and 910 grammes respectively).

For two days the animals seemed unusually quiet, but since then no untoward effects have been observed (over one month of observation).

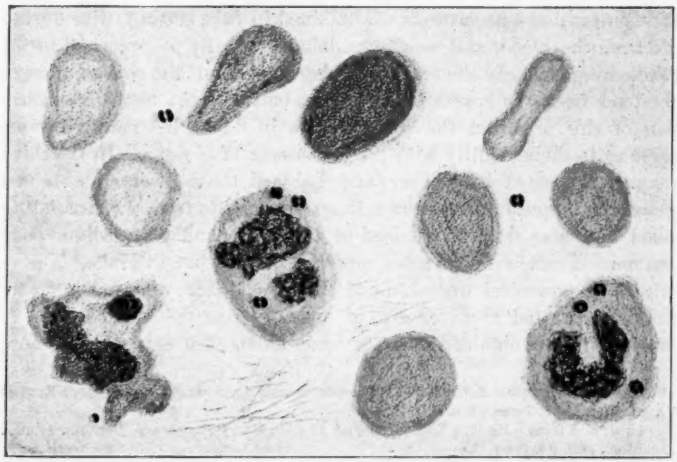
[1] Taav. Laitinen. Beitrage zur Kenntniss der Biologie des Gonococcus (Neisser), Centralb. f. Bakt. u. Parasit., 1898, vol. xxiii. p. 874. This consists of cyst or ascitic fluid, and bouillon containing 1 per cent. peptone and 0.5 per cent. sodium chloride.

FIG. 1.



Photograph of the left heart, showing the mitral vegetations. Those on the chordæ tendinæ do not show in the picture.

FIG. 2.



Cover-slip preparation from the vegetation on the valve. Stained with gentian-violet. Leitz oil immersion 1/12, ocular 5. Enlarged.

Of the remaining cultures—heart's blood, liver, gall-bladder, spleen, splenic infarct, lung, kidney, bone-marrow, and urinary bladder—those from the spleen, lung, splenic infarct, bone-marrow, and kidney remained still sterile at the end of nine days. From the liver, gall-bladder, and urinary bladder a pure culture of a moderately motile, gas-producing bacillus, which produced indol, was obtained. Tinctorially and culturally the organism behaved like the bacillus coli communis.

The cultures from the heart's blood all proved negative except in a single blood-serum tube, in which one small gray colony was observed at the end of forty-eight hours. It consisted of cocci quite similar to those already described in connection with the cultures from the valves. It also decolorized when stained by Gram's method. Transplantation upon simple agar-agar, blood-serum, and the sugar agar-agar and ascitic fluid combination were unsuccessful. The organism was lost, owing to the failure of recultivation.

Summary of Bacterial Findings. Gonococcus in the heart's blood and valvular vegetations; bacillus coli communis in the liver, gall, and urinary bladders. Remaining organs sterile.

The morphological characters and cultural behavior, taken with the tinctorial reactions of the micro-organism isolated from the blood and valvular vegetations, certainly, are so conclusive that no reasonable contention can possibly arise as to the gonorrhœal nature of the septicæmia and endocardial lesion.

The number of cases in which the evidence of the purely gonococcal nature of the complicating lesion may be considered definitely proven is so few that it seems worth while recapitulating their main features.

The first case really belonging to this group was reported by Thayer and Blumer;[1] the gonococcus was obtained in pure culture twice during life from the blood, and was found microscopically post-mortem in the affected regions. In the case of Dauber and Borst[2] the gonococcus was obtained from the heart's blood in pure culture. As to the identification of this organism the authors were in doubt, but most observers agree as to their identity with the gonococcus of Neisser. In the third instance, reported by Thayer and Lazear,[3] these observers obtained gonococci in pure culture three times during life from the circulating blood and also from the lesions of the heart and pericardium post-mortem. From a fourth case, reported by Rendu and Halle,[4] a pure culture of gonococci was obtained during life from the endometrium and demonstrated in cover-slips of exudate from the elbow-joint. A pure culture was obtained from the heart lesion after death. Still more

[1] Thayer and Blumer. Endocardite Ulcéreuse blennorrhagique. Arch. de Méd. Exp. et d'Anat. Path., 1895, Tome vii. p. 701.

[2] Dauber and Borst. Maligne Endocarditis im Anschluss an Gonorrhoea. Deutsch. Arch. f. klin. Med., 1896, Bd. lvi. p. 231.

[3] Thayer and Lazear. Loc. cit.

[4] Rendu and Halle. Bull. et Mém. Soc. Méd. des hôp. de Paris, 1897, 3 serie, Tome xiv. p. 1325.

conclusive than any is that of Lenhartz,[1] in which he obtained a pure culture of the micrococcus gonorrhœæ from the aortic valve vegetations, and, inoculating some of this into a previously healthy human urethra, incited urethritis with purulent discharge, in which organisms in every way, morphologically and tinctorially, typical of the gonococcus, were demonstrated. Finally, from my own case—the sixth—I succeeded in demonstrating microscopically the gonococcus in the urethra and valvular vegetations. I was further able to cultivate this microorganism in pure culture from the blood of the heart and mitral lesion post-mortem.

This complicating endocarditis of gonorrhœa is distinguished by no special anatomical feature from those endocarditides induced by the more common pyogenic bacteria. For the fifteen most probably purely gonococcal infections of the endocardium, Thayer and Lazear give the following table as to anatomical distribution. To this we have added our own case and that of Berg's[2]—an aortic lesion:

Left heart	Aortic,	8	Right heart	Tricuspid,	1
	Mitral,	8			
	Both,	2		Pulmonary,	2
		13–76.5 per ct.			3–17.6 per ct.

Both sides: all four valves, 1–5.9 per cent.

The symptomatology of gonorrhœal endocarditis is essentially the same as that met with in the other forms of ulcerative lesion. Complicating arthritis, when present, often precedes the heart affection; in others, as in the one which I have herein reported, the arthritic trouble may come on after the development of the cardiac lesion. Pericarditis is rare; in the case of Thayer and Lazear it was present. Councilman has reported one case in which myocardial abscess existed without any endocarditis.

Conclusions. 1. Gonococcal urethritis may be the starting-point for a fatal septicæmia induced by a pure infection with the gonococcus.

2. Endocarditis and arthritis are occasionally complications of such an infectious disease.

3. The endocardial processes may be incited by the gonococcus without the association of other organisms.

[1] Lenhartz. Ueber acute ulceröse gonorrhoische Endocarditis. Berl. klin. Woch., 1897, Bd. xxxiv. p. 1138.

[2] Berg. Pyelonephritis and Ulcerative Endocarditis as a Complicating Factor of Gonorrhœa, Medical Record, 1899, vol. lv. p. 602.

OBSTRUCTIVE BILIARY CIRRHOSIS.

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The recent occurrence in Montreal of a case of cirrhosis of the liver which seemed to stand in etiological relationship to the pressure on the common bile-duct of an enlarged gland at the hilus of the liver, the autopsy material from this case being studied at the Royal Victoria Hospital, has led to a review of the literature of cirrhosis of the liver, with special reference to the frequency of this variety.

Although it has been recognized for many years that long-continued obstruction of the bile-ducts might cause an increase of the fibrous tissue of the liver, yet obstructive biliary cirrhosis was only established as a distinct pathological condition in 1874 and in 1876 by the labors of J. Wickham Legg, of London, and J. M. Charcot, of Paris. It was due to the observations of these men more than to any other investigations that light was thrown on the confused classification of morbid conditions of the liver, and it is to their great renown that they combined pathological, clinical, and experimental evidence in favor of their views. In vol. ix. of the *St. Bartholomew's Hospital Reports*, published in 1873, there appeared Wickham Legg's first experimental observation in this field, under the title of "On the Changes in the Liver which follow Ligature of the Bile-ducts." Legg tied the common bile-duct, necessarily without antiseptic precautions, in sixteen cats, twelve of which were left for a sufficient time to obtain characteristic changes in the liver. By these experiments he produced a well-marked jaundice, great emaciation, and finally death of the animals on which he had operated, and found at autopsy an enlargement of the liver, a distinct increase of the interlobular connective tissue, and an atrophy of the liver cells.

It may be noted that a similar result had been obtained experimentally by Oscar Wyss in 1866 and by Heinrich Mayer in 1872. Legg's first clinical observations were published in the *Transactions of the Pathological Society of London*, 1874 (vol. xxv., pp. 133 and 155). He here reports two cases. In the first case, an unmarried woman, aged twenty-three years, who during her short life had been highly intemperate, and who had shown for some two weeks before death marked jaundice, at autopsy was found to have a gallstone in the common duct, a dilatation of all the bile passages, and an interlobular cirrhosis of the liver. The second case was that of a man, aged thirty-five years, with a history of strict temperance, who had been jaundiced nine months and had an

outbreak of xanthelasma multiplex on his face. At post-mortem hydatids of the liver, omentum, and recto-vesico pouch were found, with a resulting compression of the hepatic duct and an increase of the connective tissue about the portal canals. There was no other evidence of cirrhosis of the liver, and the obstruction of the hepatic duct was not complete.

In 1876, however, Legg published a case of congenital deficiency of the common bile-duct, the cystic and hepatic ducts opening in a blind sac, in which there was a well-marked interlobular and intralobular cirrhosis of the liver. In the same article he reviews the literature of congenital deficiency of the bile-ducts, and concludes that there is in all cases a cirrhosis of the liver dependent on the diseased condition of the ducts.

In the same year there appeared in the *Archives de Physiologie* Charcot and Gombault's well-known monograph on the diseases of the liver. These observers adopt an elaborate classification of the cirrhosis of this organ, in which classification, unfortunately, the terms used (annulaire, insular, and monolobulaire) are not always capable of proper interpretation. They recognize Hanot's cirrhosis, or hypertrophic cirrhosis with jaundice; Laennec's atrophic cirrhosis, and the cirrhosis dependent on obstruction of the biliary passages, the latter being anatomically identical with Hanot's cirrhosis. In the same monograph are reported four cases of cirrhosis of the liver following obstruction of the bile-ducts, the obstruction being caused by gallstones in the gall-bladder, by gallstones in the ampulla of Vater, cancer of the head of the pancreas, and angiocholitis of the larger bile passages. The anatomical picture of the livers of these cases is the same as that seen in Hanot's cirrhosis.

On the experimental side, Charcot reported observations on seven rabbits, in which he ligated the common bile-duct, producing jaundice and emaciation during life, and finding after death an enlargement of the liver, dilatation of all the bile passages, an interlobular cirrhosis, and many changes in the epithelium of the bile canaliculi. Charcot naturally operated without strict asepsis, and the presence of many leucocytes and small abscesses in various parts of the liver is a proof that he caused a serious infection of this organ; the question is thus raised whether the fibrous changes he saw in the livers of these rabbits were due simply to the damming back of bile in the dilated ducts, and its subsequent action on the liver cells, or to the growth of micro-organisms introduced at operation.

The various clinical and pathological observations on the liver were summed up in 1882 by Mangelsdorff, of Leipzig, who collected all the cases of cirrhosis of the liver which he could find recorded up to the date of publication of his own article. These cases he presented in

abstract in the *Deutsches Archiv für klinische Medicin.* He reports altogether 321 cases of cirrhosis, and of this total number 184 cases were shown to be due to obstruction of the biliary passages, eliminating, moreover, all of these cases in which a certain or doubtful history of either syphilis or alcoholic intemperance might be supposed to have any decided action on the liver itself. Mangelsdorff concludes that there is no particular variety of cirrhosis which can be said to be dependent upon this disease of the bile passages, but that practically any condition of fibrosis of the liver can follow obstruction of the ducts.

In the literature since Mangelsdorff's compilation of cases—that is, from 1882 to 1900—we have been able to find only twenty-one distinct cases of cirrhosis of the liver due to obstruction of the biliary passages, if we exclude, on the one hand, those cases in which only a partial obstruction was associated with cirrhosis of this organ, and, on the other hand, those cases in which the statements of the writers are somewhat indefinite as to the exact pathological conditions. Thanks to the kindness of Dr. Merrill and Dr. Fletcher, of the Surgeon-General's Library at Washington, who allowed us to use their complete card catalogue, we have been able to bring this historical review up to May 1, 1900, and thus to present a fairly complete list of cases for the past eighteen years. To the twenty-one cases found already reported we may add three cases taken from the records of the Royal Victoria Hospital in Montreal, which show this condition in both the early and the late stages. The following is a list of the cases on record:

Case I. (Maffucci. *Giornale Internationale della Scienze Mediche,* 1882, nouva serie, anno 4–2, Napoli, pp. 889–910).—Man; intense jaundice and ascites; liver soft on surface, olive-green, rougous, atrophied; cystic ducts dilated and containing a little liquid like egg albumen; common duct enormously dilated; hepatic duct slightly dilated. Below the common duct a band of connective tissue, which for 0.5 cm. compresses the duct. All lobules of the liver show connective tissue diffused about the parenchyma, which is diminished in volume; connective tissue between acini. Interlobular and intralobular cirrhosis.

Case II. (same author).—Man; cancer of the pancreas pressing on the common duct; intense jaundice, ascites; liver small, surface soft, green; cystic and common ducts dilated. Head of pancreas transformed to a carcinomatous tissue, which completely occludes the common duct; all bile capillaries dilated. Interlobular and intralobular cirrhosis.

Case III. (same author).—Woman; intense jaundice, ascites; carcinoma of stomach, with many secondary nodules in the peritoneum, one compressing the common duct at the point of union of cystic and hepatic ducts; hepatic and common ducts dilated; liver olive-green, atrophied, surface smooth, soft; all bile-ducts dilated and containing bile. Interlobular and intralobular cirrhosis of the liver.

Case IV. (Heneage Gibbs. *Transactions of the Pathological Society of London,* 1883, vol. xxxiv., p. 129).—Male infant; family history

good; born December 3, 1881; average development; jaundiced skin and conjunctiva a few days after birth; persistent clay-colored stools, emaciation, ascites; death July 10, 1882. At post-mortem, liver hard, smooth, weight 4 ounces; no common duct; occlusion of pancreatic duct; hepatic duct filled with fibrous mass; interlobular connective tissue much increased. Both interlobular and intralobular cirrhosis of the liver.

CASE V. (Henry Ashby. *Archives of Pediatrics*, 1884, vol. i., p. 537).—J. H., admitted to the Dispensary for Sick Children at Manchester, October 4, 1883. Family history negative; maternal, syphilis. Jaundice since birth; urine bile stained; stools clay-colored; liver enlarged, edge at umbilicus. Death December 30, 1883, aged four and a half months. At post-mortem all organs bile stained; no ascites; liver seven ounces, not enlarged, dirty-green color, surface granular, tough feel, creaking when cut. On section, dark-green islets of fibrous tissue, especially about the portal vessels. Small cystic duct entering the common duct and pervious to duodenum; no hepatic duct; bile-duct ending in fibrous tissue at the transverse fissure; microscopically fibrous tissue about the portal vessels. Both interlobular and intralobular cirrhosis.

CASE VI. (Alivia. *Contributo alla Casuistica della Cirrhosi Biliare*, Sassari, 1885).—Patient admitted November 13, 1883. Tumor in right hypochondrium, painful, fluctuating in size; pain in right hypochondrium, passing to epigastrium; nausea, vomiting, diarrhœa, jaundice; dark-colored urine; no clay-colored stools; irregular fever; death. At autopsy, perihepatitis, hepatic cirrhosis, biliary calculi, dilatation of bile channels, two gallstones in the gall-bladder.

CASE VII. (Michael. *Archives of Pediatrics*, 1885, S. 573; also *Archiv für Kinderheilkunde*, vii., 2, S. 137. Quoted by Gese).—Boy; jaundice since birth; clay-colored stools; dark-colored urine; great emaciation; death, second month. Liver not enlarged, green, creaks on cutting, strands of fibrous tissue through it; larger bile channels filled with bile; common duct ends in fibrous tissue.

CASE VIII. (Gessner. *Ueber Cong. Verschluss der Gallenausfuhrungsgange*, Halle, 1886. Quoted by Gese).—Boy; jaundice since birth; urine contains bile; feces colored; vomiting, cough, emaciation. Death, third month. Liver enlarged, grayish-green, hard, surface granular; connective tissue increased; cystic, hepatic, and common ducts obliterated except for pinpoint lumen in the common duct.

CASE IX. (Legry. *Bulletin de la Société Anatomique de Paris*, 1889, Anne 64).—E. C., female, May 9, 1889; aged fifty-seven years. At twenty years, in the sixth confinement, jaundice, pain in right hypochondrium and epigastrium; no clay-colored stools; chronic jaundice, œdema, and ascites; collateral circulation developed; urine quite dark. Post-mortem May 22d. Liver enlarged, surface granular, very hard, cuts with difficulty; bile channels dilated, contain biliary concretions; glands generally enlarged. An interlobular and an intralobular cirrhosis.

CASE X. (Cnopf. *Münchener medicinische Wochenschrift*, 1891, Nos. 16 and 17. Quoted by Gese).—Child; jaundice second day; white stools; urine containing bile-pigment. Death, tenth week. Liver normal in size, smooth surface; jaundiced interacinous and intraacinous tissue increased; gall-bladder and duct quite lacking.

CASE XI. (Cnopf. *Münchener medicinische Wochenschrift*, 1891, Nos.

16 and 17. Quoted by Gese).—Child; three days jaundiced; urine icteric; grayish yellow, thin stools; emaciation; ecchymoses on face and neck; liver enlarged, sensitive; bloody diarrhœa. Death, thirty-third week. Liver enlarged, cirrhotic.

Case XII. (Rolleston and Kanthack.[1] *Archiv für klinische Medicin*, 1892, p. 488).—Child, one month old; jaundiced; cystic disease of liver and kidney, with hypertrophic ciliary cirrhosis; bile passages much dilated, connective tissue about them much increased; great increase of bile-ducts; vacuolization of liver cells. Cirrhosis of liver; no definite obstruction of ducts.

Case XIII. (Parkes Weber. *British Medical Journal*, April 25, 1895, p. 1027).—Mrs. M. S., aged fifty-five years; jaundice four years; pain in left side; no hepatic colic, no children, no miscarriages, no syphilis, no alcohol (?); xanthelasma palpebrarum. Liver hard, uniformly enlarged; œdema of feet and ankles; dulness of liver from fifth rib to level of umbilicus; pruritus; later, ascites; liver not to be felt; stools colorless or mixed with blood; urine contains bile; no dilatation of abdominal veins; temperature 100° F. Coma and death, December 24, 1895. At post-mortem, liver 54 ounces, tough, surface puckered or granular, green; gall-bladder dilated, hypertrophied, contains green fluid; cystic and hepatic ducts pervious, common duct dilated; two black gallstones in ampulla of Vater, size of cherry-stones; no ulceration; contents of gall-bladder could be squeezed into the duodenum.

Microscopical Examination. Multilobular cirrhosis; no multiplication of smaller bile-ducts (liver presents ordinary cirrhosis, type of hypertrophic cirrhosis due to gallstones). Author compares Charcot's case, *Archives de Physiologie*, second series, vol. iii., p. 295.

Case XIV. (Chadwick. *British Medical Journal*, 1895, vol. i., p. 1143).—Woman, aged thirty-six years; copious hæmatemesis; jaundice and gallstones six years; atrophy of liver; xanthelasma; liver green, much cirrhosed, fibrous tissue like ordinry cirrhosis.

Case XV. (Gese. *Jahrbuch fur Kinderheilkunde und physiche Erziehung*, N. F., 1896).—Twenty-five cases of occlusion of the bile-ducts, with a number of cases of cirrhosis of the liver.

One new case reported: Child; jaundiced eighth and ninth day; clay-colored stools; urine containing bile-pigment; liver enlarged. At autopsy, obliteration of hepatic duct; patent common duct; open gall-bladder, fluid from gall-bladder passing into duodenum; liver cirrhotic; cirrhosis irregular, both interlobular and intralobular in type.

Case XVI. (Stevens. *Archives of Pediatrics*, 1896, vol. xiii., p. 733).—Wm. McA., aged four months; family history negative; jaundice at birth; stools clay-colored; urine clear, green; later, blood in vomitus and stools, which became black and tarry. Death from hemorrhage. At autopsy, absence or obliteration of hepatic and cystic ducts; cirrhosis of liver; hemorrhage into stomach and intestines; deep jaundice; cystic, hepatic, and common ducts transformed to fibrous cords,

[1] In the above case of Rolleston and Kanthack no definite obstruction of the bile passages could be detected. Nevertheless, the dilatation of the biliary channels was so marked and the type of cirrhosis was so similar to that seen in the cases in which a positive obstruction could be demonstrated, as was shown by the illustration in their paper, as to make it quite proper to include their case in this list of cases of true obstructive cirrhosis.

absolutely impervious. Liver large, dark green on section, marbled, free surface granular.

Histological Examination. Cirrhosis of liver, involving groups of lobules (multilobular, possibly interlobular). Bile-ducts much increased.

CASE XVII. (Kynoch. *Edinburgh Medical Journal*, 1896, vol. xvii., pp. 35–37).—Matilda C., aged three months and four days, admitted to Dundee Royal Infirmary at two and a half months; jaundice since birth; skin and conjunctiva green color; emaciation; abdomen distended; liver enlarged, edge sharp; no ascites. At post-mortem, liver 237 grammes (8 ounces), edges sharp, green, tough in consistence, surface irregular, fibrous bands of yellow color; common duct, at junction of cystic and hepatic ducts, narrow, thread-like, just pervious to bile. Microscopically, slight biliary cirrhosis.

CASE XVIII. (Krokiewiez. *Wiener klinische Wochenschrift*, 1898, pp. 321–323).—M. S., aged fifty-seven years; jaundice; anorexia; constipation; pain in abdomen one year; chills at onset; œdema of legs, emaciation, and ascites; liver enlarged, painful, border 12 cm. below right costal margin, edge hard and thick, surface smooth; urine bile stained; tumor in region of gall-bladder; jaundice one year; ascites. Coma and death, August 14, 1897. At post-mortem, dilatation of common, hepatic, and cystic ducts; stenosis of common duct. Liver 3 cm. below costal margin, surface smooth, edge broad, capsule thickened, parenchyma hard, greenish-brown; bile-ducts dilated; gall-bladder enlarged, wall thick, contains few gallstones. Interlobular cirrhosis of liver; stenosis of common duct.

CASE XIX. (Boinet. *Archives Générale de Médecine*, 1898, vol. i., p. 385).—W., aged thirty-five years; alcoholic, non-syphilitic; gastro-intestinal catarrh and dysentery. First seen in 1894; jaundice six years; xanthelasma palpebrarum on both sides; urine high-colored; stools not clay-colored; for two and a half years liver constantly increased in size, reaching from the fifth intercostal space to within 4 cm. of crest of ilium; no ascites; no dilatation of abdominal veins. In 1896, extreme emaciation. Death from tuberculosis. At post-mortem, liver large, smooth, 4720 grammes (not hobnailed); lymphatic glands all enlarged, especially about hilus of liver, where they press on the common duct; liver cirrhotic.

CASE XX. (Rabé. *Cirrhose Periportal avec Calcul Enclave dans l'Ampoule de Vater. Mort par Pylephlebite Obliterante*, February, 1898).—Man, aged seventy-three years. Symptoms of atrophic cirrhosis and ascites. Liver 7 cm. in mammary line; contracted; dilated abdominal veins, jaundice of skin and conjunctiva; urine deep yellow; emaciation, œdema of legs and scrotum, constipation, anorexia; stools normal. Death from marasmus after five months. At post-mortem, 1200 c.c. of abdominal fluid; liver, type of Laennec's cirrhosis, very hard, chamois-gray, surface granular; hepatic, cystic, and common ducts dilated; calculus in common duct, another in ampulla of Vater. Periportal and interlobular cirrhosis, with great increase of bile-ducts.

CASE XXI. (Benner. *Ein Fall von Gallenstauung-cirrhose der Leber nach primaren Adenocarcinom im Ductus Choledochus.* Inaugural Dissertation, 1899).—Post-mortem, typical cirrhosis of the liver. Dilatation of bile-ducts: adenocarcinoma of common duct; retention of bile in duct. Interlobular cirrhosis about ducts; jaundice of all organs; great dila-

tation of all small bile-ducts. Liver enlarged and cirrhotic, weighing 2210 grammes.

Case XXII. (hitherto unreported).—Private case occurring in the practice of Dr. D. J. Evans, of Montreal. Male child, born of healthy parents, November 29, 1897. Breast-fed; digestion always bad; stools greenish; eczema capitis at three months of age. One month later, tuberculous glands of neck, caseous and suppurating. At a year and a half child became fretful and irritable; urine high colored; stools green; abdomen somewhat increased in size. One month later jaundice of conjunctivæ, clay-colored stools, great enlargement of abdomen, hypertrophy of liver, edge at crest of ilium; enlargement of spleen; later, contraction of liver, surface rough and lobulated. Development of œdema of extremities; congestion of abdominal veins, ascites, wasting, and emaciation; skin lemon-yellow in color; many petechiæ over palms, neck, and chest. Abdomen tapped and 24 ounces of clear, yellowish fluid removed. Collection of ascitic fluid almost immediately; emaciation and wasting extreme; great cachexia. Death, July 14, 1899.

The autopsy was performed by Dr. Anderson, of Montreal, examination of abdominal organs only permitted. Deep jaundice of skin and conjunctivæ, numerous petechiæ of skin of abdomen; large amount of ascitic fluid; pale yellow in color. Great hypertrophy of spleen; enlarged inflammatory gland at hilus of the liver pressing on the common duct, completely obliterating its orifice; liver greatly enlarged, surface made up of large lobules of liver substance lying between dense, firm bands of connective tissue, surface very rough. On section, liver cuts with difficulty, showing a great increase of fibrous tissue, which lies in islands between the hepatic lobules. Interlobular and intralobular cirrhosis of the liver.

Microscopical Examination of Liver. The capsule is thickened, with some infiltration of small round cells. Just underneath the capsule is a layer of fibroblasts, the protoplasm of which stains lightly with eosin, and the spindle-shaped nuclei stain deeply with hæmatoxylin. From the capsule the connective tissue has extended throughout the whole organ, lying in broad bands between the hepatic lobules, which are thus marked off into smaller or larger areas of normal liver substance. The strands of new tissue are made up of well-developed fibres and young fibroblasts. At the periphery of the organ bands of tissue join the capsule almost at right angles, and there spreading out into broad bundles round off or top off the hepatic lobules.

In the portal spaces the fibrous tissue has developed concentrically about the bloodvessels and bile-ducts, which are considerably dilated. Close to the vessels the tissue forms a ring of well-developed, rather lightly staining, fibrous tissue, in the periphery of which, where the process is less advanced, may be seen a layer of fibroblasts. These fibroblasts are especially abundant, extending everywhere, from the older deposit of fibrous tissue into the liver lobules, isolating and surrounding groups of liver cells. At times these fibres spread out between the hepatic elements, giving an appearance much like the branching of a tree. Throughout all the lobules, moreover, between the individual liver cells, may be seen isolated fibres of connective tissue—the cirrhosis is thus both *interlobular* and *intralobular* in type.

Coincidently with the deposit of fibrous tissue definite changes have occurred in the liver cells; near the centres of the lobules the cells are

normal in size, the protoplasm is well preserved, and the nuclei are lightly stained with hæmatoxylin. At the periphery the cells are atrophied and compressed by the fibrous tissue, the protoplasm has almost entirely disappeared, and the nuclei are greatly shrunken. The liver cells thus are in a state of chronic inflammation, or, more properly, have degenerated to their earlier epithelial type. Where the layer of fibroblasts is especially abundant the liver cells are still more atrophied, there remaining practically only the nuclei, which are lined up in tubules or cords and surrounded by the new tissue. These nuclei are somewhat compressed and stained a little more deeply than the normal nucleus of the liver cell. These *cords* or *tubules* of cells, formed of little more than nuclei, are the elements which have been called *bile-ducts*, *bile capillaries*, or more recently *intermediate* or *pseudo-bile-ducts*.

The source of origin of these reduplicated bile-ducts has been in doubt for some years. It is maintained, on the one hand, that these ducts originate from the reduplicated epithelium of the older bile-ducts, while, on the other hand, their origin from compressed atrophied liver cells has been especially emphasized within the past decade. Findlay, who uses the term *intermediate* or *pseudoducts*, considers that they are simply liver cells which have degenerated and assumed the character of primitive hepatic elements, and later come to be connected with the normal bile-ducts. In the present case the origin of these reduplicated bile-ducts from liver cells is readily seen. It is possible to trace all the stages from the normal liver cells to the chronically inflamed liver cells with deficient protoplasm, and then to the rows of liver nuclei lined up in cords or tubules between the layers of fibrous tissue, and presenting an appearance quite similar to that of normal bile-ducts.

This reduplication of bile-ducts has reached an extreme grade in this case; they are especially abundant at the edge of the lobule in the zone of fibroblasts, where the new tissue seems to be surrounded by a wreath of ducts, outside of which lies the normal hepatic parenchyma. This wreath-like arrangement is observable in all the portal spaces; it seems more marked in this disease than in any other pathological conditions of the liver, and may possibly form a histological feature characteristic of obstructive cirrhosis and differentiating it from other varieties of cirrhosis of this organ. (See Fig. 1.)

The strands of new tissue are infiltrated with small round cells, this infiltration being especially rich in the layer of fibroblasts, where the wreath of bile-ducts is to be made out. There are many small round cells as well scattered irregularly through all the new fibrous tissue and deposited between the liver cells in the centres of the lobules; in some areas the strands of new tissue are so infiltrated with small round cells as to suggest an inflammatory origin for the fibrous tissue present.

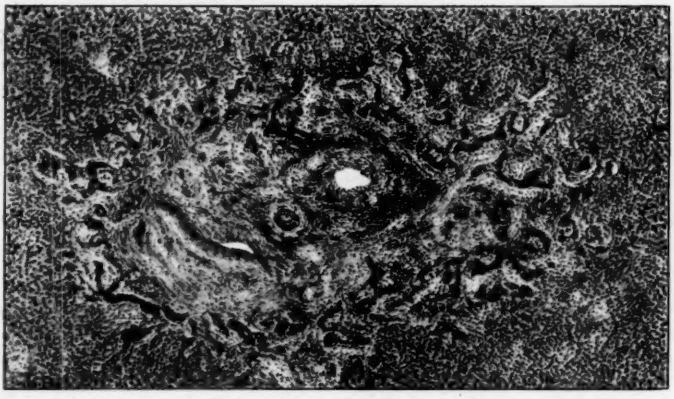
The walls of the larger bloodvessels are thickened and their lumen filled with blood cells. The smaller bloodvessels are occasionally dilated and in a state of acute inflammation, about the vessel wall being seen a deposit of round cells, with a few connective tissue nuclei. The central veins of the lobules are everywhere dilated; the lobules themselves are congested, often the red blood-corpuscles being massed together in large numbers between the hepatic elements.

The bile channels are dilated and full of brownish-black bile pigment, which is deposited irregularly throughout the bile passages of the whole organ. As a rule, this deposit of bile is most marked about the centres

of the lobules, where often the liver cells are themselves stained yellow with the liver secretion. A similar precipitate of bile-pigment may be observed in the portal spaces and at the edges of the lobules.

Aside from the precipitate of bile-pigment, in many areas of this liver may be seen fine, black granules, coccoid or diplococcoid in shape, which are deposited both intracellularly and extracellularly. The interpretation of the nature of these bodies is exceedingly difficult unless one considers that they are micro-organisms in the progress of disintegration. This view is strengthened by the appearance of the bile-ducts in this case, which are in a state of active inflammation, their lumen being filled with well-formed bacilli. An etiological relationship between these granules and the new tissue developed in this liver was suggested by this appearance, but such a relationship was not confirmed by the examination of later cases of obstructive cirrhosis.

FIG. 1.



Specimen hardened in Müller's fluid, embedded and sectioned in paraffin stained with hæmatoxylin and eosin. Magnification 105 diameters. Between the hepatic lobules surrounding the portal vessels is seen the new-formed fibrous tissue in concentric rings, at the periphery of which lies the wreath of reduplicated bile-ducts. Slight infiltration with leucocytes. Brownish deposit of bile pigment in the liver parenchyma. Interlobular and intralobular cirrhosis.

CASE XXIII. (hitherto unreported).—Maud V., aged twenty-eight years, admitted to the Royal Victoria Hospital, November 25, 1898; died December 22, 1898.

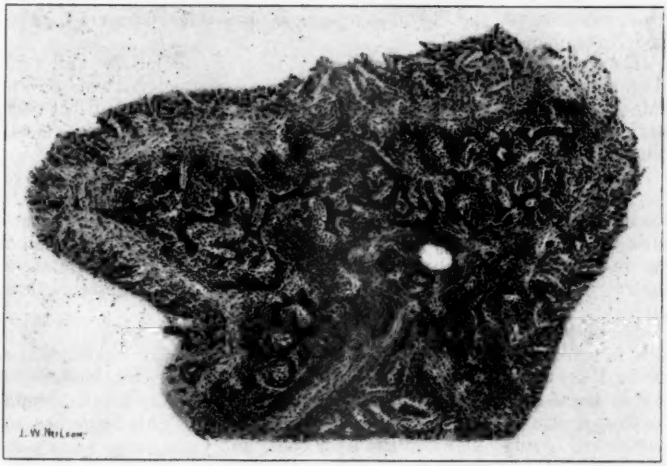
History. Jaundice three years, increasing in intensity; pruritus of skin; clay-colored stools; tenderness in right hypochondrium; anorexia; vomiting, vomitus greenish in color; œdema of the legs; dilated abdominal superficial veins; ascites; hydrothorax; coma and death.

At post-mortem, liver greatly enlarged, weighing over 4000 grammes, surface granular; liver firm, on section cutting like leather; gall-bladder thickened; cystic duct obliterated; hepatic duct slightly dilated, containing a loose calculus pressing on the cystic duct at its orifice. Interlobular and intralobular cirrhosis of the liver.

Microscopical Examination. The morbid changes seen in this liver are quite similar to those seen in the preceding case, but somewhat less advanced in their development. Between the hepatic lobules there are broad bands of tissue, made up of well-developed connective tissue fibres, in the periphery of which are many fibroblasts which penetrate the lobules between the liver cells, isolating and surrounding individual groups of cells. The cirrhosis is both interlobular and intralobular in character.

In the portal spaces where the tissue has developed in concentric rings about the bloodvessels and the bile channels one sees a zone of lightly staining, well-formed fibrous tissue, surrounded by a layer of fibroblasts, in the periphery of which lies a wreath of reduplicated bile-ducts. Outside this wreath lies the normal hepatic parenchyma. This wreath-like arrangement is visible in this liver in many of the portal spaces, but more frequently between the hepatic lobules the bile-ducts are lined up at right angles to the strands of fibrous tissue. (See Fig. 2.)

FIG. 2.



Specimen hardened in Müller's fluid, embedded and sectioned in paraffin stained with hæmatoxylin and eosin. Magnification 70 diameters. Moderate development of fibrous tissue between and within the lobules. Considerable infiltration with leucocytes. Wreath-like arrangement of reduplicated bile-ducts. Interlobular and intralobular cirrhosis.

In the centres of the lobules the liver cells are still preserved, but at the edges of the lobules they are atrophied and compressed. One sees the same transition from the normal liver cells to the chronically inflamed liver cells and thence to the reduplicated bile-ducts; there is no vacuolization and no fatty degeneration of the liver cells.

The walls of the larger bloodvessels are slightly thickened, the smaller bloodvessels being somewhat dilated; the central veins are especially enlarged and the liver lobules are markedly congested throughout,

many red blood-corpuscles lying free among the hepatic elements. The escape of blood in some cases has been so marked as to suggest capillary hemorrhages.

The bile channels are not dilated; there is no special deposit of bile pigment, and only a few liver cells are seen which are slightly stained with bile.

The infiltration with leucocytes is very marked, especially in some of the strands of new tissue, where the deposit of both polymorphonuclear and small mononuclear leucocytes is so extensive as to again suggest an inflammatory origin for the tissue changes. This infiltration, moreover, is especially abundant about the new bile-ducts, but in the lobules themselves, between the liver cells, are often found many isolated small round cells.

The characteristic appearances of biliary cirrhosis are thus seen in this case as well as in the preceding one, and while the presence of leucocytes is strongly suggestive of inflammation, yet there are no bacteria present in any of the bile-ducts and no especial deposit of coccoid or diplococcoid bodies in or between the liver cells.

CASE XXIV. (hitherto unreported).—Walter B., aged fifty-three years, admitted to the Royal Victoria Hospital, November 14, 1899; died November 19, 1899.

History. Attacks of pain in the epigastrium, tenderness, and vomiting, over a period of two years; jaundice; clay-colored stools; bile-stained urine; enlargement of liver and tenderness over gall-bladder; marked itching of skin. Operation: cholecysduodenostomy. Death from hemorrhage.

At post-mortem, liver yellow, granular, firm, and enlarged, on section cutting with difficulty, showing islands of fibrous tissue; cystic, hepatic, and common ducts greatly dilated; ulceration about the ampulla of Vater, especially on the duodenal side, where there is a slight tear in the mucous membrane and an ulcer about the bile papilla, showing recent passage of gallstone; no calculus found at operation or at autopsy. Interlobular cirrhosis of the liver.

Microscopical Examination. (See Fig. 3.) The histological changes seen in this liver are indicative of an early stage of cirrhosis, dependent upon obstruction of the bile passages. The development of fibrous tissue is little marked, but between the lobules may be seen a number of strands of thickened connective tissue. This tissue has not encroached to any extent on the liver cells, and there seems to be comparatively little development of fibrous tissue in the lobules themselves. The tissue itself is largely made up of fibroblasts, with spindle-shaped nuclei. This tissue is specially well developed about the portal spaces, where it is very rich in nuclei. In a few of the portal spaces a concentric arrangement of this tissue about the portal vessels and the dilated bile-ducts may be observed, and a similar zone of fibroblasts, with a wreath of reduplicated "bile-ducts," is seen about the older fibrous tissue. The number of these bile-ducts is quite small, however, and the wreath-like arrangement is only observable in a few areas. The infiltration with leucocytes is especially great in the strands of new tissue; in many of the liver lobules there is a rich deposit of leucocytes among the liver cells, especially at the edge of the lobules.

The bloodvessels are everywhere dilated, both in the portal spaces and in the centres of the lobules. The walls of these vessels are con-

siderably thickened and their lumen filled with red blood-cells. About the central veins there is a rich deposit of fibrous tissue infiltrated with mononuclear leucocytes; here the liver cells are greatly atrophied and have in many cases quite disappeared. In the periphery of the lobule as well the liver cells are shrunken and chronically inflamed.

FIG. 3.

Specimen hardened in Müller's fluid, embedded and sectioned in paraffin stained with hæmatoxylin and eosin. Magnification 70 diameters. Deposit of fibrous tissue between the liver lobules. Marked infiltration, with leucocytes. Moderate reduplication of bile-ducts, which show the wreath-like distribution. Interlobular cirrhosis.

The bile-ducts are everywhere dilated and full of bile. A deposit of bile-pigment has taken place in many of the liver cells about the central veins of the lobule, where often these cells are stained yellow with this pigment. A radial distribution may often be made out about the dilated central veins.

The microscopical changes in this case reveal a cirrhosis of the liver, interlobular in character, the fibrous tissue having developed about dilated bile channels. The dilatation of these channels and the deposit of bile-pigment is so great as to indicate a long-continued obstruction to the flow of bile, such an obstruction evidently being the etiological factor concerned in causing the cirrhosis.

Analysis of the Above Cases.

Causes of Obstruction. *Congenital Deficiency of Bile-ducts.* The most frequent cause of complete obstruction to the flow of bile is the congenital deficiency of the hepatic and cystic ducts, these ducts terminating in blind, impervious cords. The livers of these congenital cases are enormously enlarged, the edge often reaching to the anterior-superior iliac spine, the enlargement either being observed at birth or taking place within a few weeks after the entrance into life. The children subject to this disease develop deep jaundice, profound cachexia, emaciation, ascites, œdema of the extremities, and various cutaneous disorders. They usually die at the age of six or eight weeks.

At autopsy the livers are found to be greatly enlarged, with a rough, hard surface. On section they cut with difficulty, show dilated bile channels full of bile and islands of connective tissue. Microscopical examination reveals an interlobular cirrhosis of the liver of an extreme grade, with great reduplication of the smaller bile-ducts. Death intervenes in these cases within a few weeks after birth, and in spite of the extreme and persistent jaundice which one observes a suspicion arises that the retention of bile is not the cause of the cirrhosis of the liver, but that the increase of the fibrous tissue in this organ is the result of the same intra-uterine morbid change which leads to the obliteration of the ducts. It must be remembered, however, that the liver is probably an active functional gland after the third month of pregnancy, and we thus have a lapse of time sufficient for the cirrhosis to develop in consequence of the obliteration of the ducts alone and the subsequent damming back of bile and its action on the liver cells without calling to our aid in explaining this condition any unknown etiological factors.

Gallstones. Gallstones stand next in order of frequency to the cases of congenital deficiency of the ducts. Here a complete obstruction to the flow of bile may be caused by the filling of the gall-bladder by a number of stones, by the lodgement of a single stone in one of the ducts or in the ampulla of Vater, or, more frequently still, by the contraction of scar-tissue which has developed about an ulceration caused by the passage of a sharp-edged gallstone. In some one of these ways the flow of the liver secretion is entirely shut off, and at autopsy the livers of these patients show a cirrhosis interlobular in character and usually moderate in extent. If the obstruction be of long standing a more advanced state of cirrhosis may be developed, the fibrous tissue developing both between and in the hepatic lobules.

It has been stated by some writers that in gallstones cirrhosis of the liver seems to develop more frequently in connection with rough, sharp-edged stones than in connection with round, smooth stones, which are less capable of causing inflammation and ulceration of the biliary passages.

Cancer of the Head of the Pancreas occurs rarely as a cause of obstructive biliary cirrhosis. Several cases have been reported, however, of this variety, either by Mangelsdorff or in the list of cases quoted in this article. The growth of cancerous tissue in the pancreas completely obliterates the lumen of the common duct, and thus causes a complete obstruction to the flow of bile, inaugurating the same series of morbid changes in the liver as is seen in the other cases.

Enlarged Glands at the hilus of the liver exerting a pressure on the common or the hepatic duct in rare instances may block the liver secretion sufficiently to cause cirrhosis of this organ. This is the condition seen in one of the cases studied at the Royal Victoria Hospital. The glands may be cancerous, tuberculous, or simply inflammatory in nature, and the livers of these cases may show both the presence of metastatic tumor nodules and an increase of the fibrous tissue about the dilated bile channels between the lobules of hepatic cells.

Macroscopical Appearance. The livers in these cases of obstructive cirrhosis are greatly enlarged, with a rough, jaundiced surface, and considerable perihepatitis. The weight of the liver is much increased, the organ hard, cutting with great difficulty, and showing on section greatly dilated biliary passages and islands of new connective tissue.

In the later stages the contraction to which all cirrhotic livers are probably liable takes place in this variety of cirrhosis in a large proportion of cases, and, in fact, these livers often approach more nearly the type of cirrhosis seen in the atrophic liver of Laennec than they do the enlarged hypertrophic organ of Hanot. The development of ascites, œdema of the extremities, and the caput medusæ of abdominal veins in many cases indicates the great obstruction to the flow of blood in the portal bloodvessels caused by the large amount of fibrous tissue present in the liver.

Microscopical Examination. Careful histological study reveals dilated bile channels, dilated bloodvessels, an increase of fibrous tissue about these dilated channels, and a cirrhosis of the liver which may be either interlobular, intralobular, or pericellular in type. As has already been indicated, groups of cells or individual cells are often seen isolated and surrounded by fibrous tissue. The presence of new bile-ducts is noted in nearly every careful anatomical description, although the wreath-like distribution of bile-ducts has not been emphasized in any cases except those studied in Montreal. It is impossible as yet to say whether this wreath-like distribution is peculiar to this variety of hepatic cirrhosis, but its occurrence in all three of the cases here reported leads to the suggestion that it may be characteristic of this disease, differentiating it histologically from other morbid conditions of the liver.

I use the term bile-ducts here to indicate those smaller bile channels lined by cubical epithelium as distinct from bile capillaries lying between the liver cells and lined by flat epithelium. The presence of these reduplicated bile-ducts cannot be considered diagnostic of any variety of cirrhosis of the liver, as they have been described in practically all varieties of this disease, in hydatids of the liver, and in certain intoxications. Wagner and Liebermeister have noted them in cases of gallstones, in alcoholic cirrhosis, and in chronic obstruction of the hepatic veins; they have been described in liver abscesses, in the syphilitic liver of Friedländer, and in chronic tuberculous peritonitis. Podwyssozki has observed them in regenerating liver tissue, and it has recently been shown by Neisser, of Leipzig, that the experimental injection of phenol solution and substances like phosphorus into the circulation is followed by the appearance in the liver of these new-formed bile-ducts.

Ackermann has shown by injection that these columns or tubules of cells described under the name of ducts are in reality bile channels, for he has been able to trace a direct connection between them and the larger bile passages.

Recently, Findlay has called renewed attention to the mode of formation of these new bile-ducts, regarding them as simply liver cells, degenerating and reverting to their embryonal or epithelial type and later coming to be connected with the older bile passages. He proposes to name them *intermediate* or *pseudo-bile-ducts*. Although their origin from chronically inflamed liver cells has been specially emphasized within recent years, and although this mode of origin is quite clearly indicated in the cases studied in this report, it must be remembered that there are many pathologists who still maintain that these new ducts are formed by the simple reduplication of the epithelium of the older bile-ducts.

Clinical Symptoms. In studying the histories of these cases of obstructive cirrhosis one is impressed by the great similarity which the symptoms of the different patients bear to each other, such a similarity being so marked as to reveal a symptom-complex and a cycle of clinical changes which are quite characteristic of a complete obstruction to the flow of bile and the morbid changes subsequent to this obstruction. The history of any one case, in fact, is repeated in the histories of practically all of the other cases.

The onset of the disease is sudden, the first symptom being an extreme jaundice, coincident with which is the appearance of clay-colored stools, which indicate the complete stoppage to the flow of bile and the retention in the blood and lymph of the patient of this liver secretion. Enlargement of the liver follows immediately, often reaching an extreme grade, the edge being found at the anterior iliac spine. The systemic disturbance now becomes marked, due doubtless to the incom-

petence of the liver and absorption of the liver secretion. The headache is painful and severe, often intermittent in occurrence; the anorexia is complete, so that the patient may be able to take no food whatever, the vomiting being frequent and exhausting, the ejecta a greenish detritus or bile-stained gastric secretion. With the appearance of the gastro-intestinal changes the various forms of cutaneous disorders develop, especially xanthelasma and subcutaneous hemorrhages. The itching of the skin is extreme, so great as to require large doses of morphine as a sedative. Fever is rare, and in fact does not occur in the cases of persistent obstruction to the flow of bile, it usually being seen in allied cases of an intermittent obstruction caused by the lodgement of a calculus in the ampulla of Vater, when usually paroxysms of chills, fever, and sweating develop, followed by partial relief of the jaundice. The emaciation and wasting are very rapid, the loss of strength and flesh taking place within a few weeks after the onset of the first symptoms.

The condition in which these various symptoms are included has been described as cholæmia, but a better definition is found in its description as *hepatic incompetence* and *auto-intoxication*. As a result of the complete stoppage to the flow of bile and the retention of this substance by the system, the liver is permanently put out of function, the retained products of digestion, instead of being further broken up and eliminated, are, together with the liver secretion, absorbed by the general circulation, and act through the blood and lymph on all the different organs and tissues of the body.

Following the enlargement of the liver we have a subsequent contraction of this organ, which occurs with great rapidity, and the appearance of which ushers in the chain of symptoms which quickly results in death. The size of the liver greatly diminishes, the fibrous tissue developing in it blocks the portal circulation and causes the dilatation of the abdominal veins (during the establishment of a compensatory circulation), œdema of the extremities, and abdominal ascites. The occurrence of the last three symptoms is common to practically all of these cases of obstructive cirrhosis, and, as we shall point out below, they are symptoms differentiating this variety of disease from the ordinary cirrhosis of the liver in which jaundice is present.

The patients subject to this disease suffer from the greatest weakness and emaciation, unable to retain food in consequence of the persistent vomiting, developing marked cutaneous disorders, and in the late stages œdema and ascites, quickly sink into a profound coma or a toxic condition, the end of which is seen only at the fatal exit.[1]

[1] It is difficult to explain the statements of several writers who allude in their description to clay-colored stools and bile-stained vomitus: the presence of bile in the gastric secretion may be accounted for, if we assume a secretion of this substance by the walls of the stomach, as

General Considerations. In considering these cases of cirrhosis of the liver associated with complete obstruction of some one of the ducts of this organ and subsequent damming back of bile on the liver cells several problems naturally present themselves:

Can obstruction to the flow of bile cause in man and animals a cirrhosis of the liver, and by what means is such a cirrhosis caused?

Is the type of cirrhosis associated with obstructions in man to be differentiated on pathological and clinical grounds from other varieties of cirrhosis of this organ?

Let us now consider those questions in their various aspects.

As we have indicated above, many experiments have been performed which seem to show that cirrhosis of the liver is in *animals* a direct consequence of ligature of the common bile-duct or its branches. In the earlier experiments, however, although the increase of fibrous tissue was always found to follow this ligature of the ducts, yet the operators conducted their experiments without modern methods, and in many cases infected their animals at the time of operation. It is noticeable in nearly every series of cases reported that a large proportion of animals died from sepsis, and it thus was impossible to decide whether the cirrhosis seen was a consequence of the damming back of bile caused by the ligature of the ducts or followed the infection of the liver and the bile passages by micro-organisms introduced at operation.

Beside the work of Legg and Charcot, already referred to, a number of pathologists have worked in this field at different periods.

Maffucci, of Naples, reported a series of experiments in which he ligated the common duct in a number of animals which lived about one month and showed at autopsy a typical interlobular cirrhosis.

Julius Steinhaus, who operated on guinea-pigs, found after ten days dilated bile-ducts and necrotic areas in the liver cells, but found no increase of bile-ducts and no increase of connective tissue in the liver. A lapse of ten days between the operation and autopsy cannot be considered as sufficient time for any great amount of cirrhosis to develop in connection with obstruction, and the failure of Steinhaus to find it is not strong evidence of a negative character.

The recent work of Vaughan Harley is quite conclusive from the experimental stand-point in answering the question whether obstruction does or does not cause cirrhosis of the liver. With the most perfect antiseptic technique Harley ligated the common duct or its branches in a number of animals, which he allowed to live for a considerable time after operation, either killing them at a late date or waiting until death

the blood and lymph channels are naturally highly charged with it. It must be remembered, however, that many of the observations of earlier writers as to the presence of bile in the vomitus were made simply from the visual appearances of the ejecta, and not from careful chemical tests.

should ensue from the morbid changes caused by the obstruction. In practically every case Harley found a definite increase of connective tissue in the liver about the bile-passages—that is, a true interlobular cirrhosis—moreover, there was no evidence that he had at any time infected the liver or any of the bile channels.

It is interesting in this connection that Nicoti and Richaud have described in dogs a condition of biliary cirrhosis of the liver dependent on the presence of gregarines in the branches of the hepatic duct. Complete obstruction of these branches is thus caused, with the resulting dilatation of the ducts behind this obstruction and a growth of connective tissue about these dilated passages. There are numerous new-formed bloodvessels and new-formed bile-ducts in the connective tissue.

Macroscopically the livers are enlarged and cirrhotic, cutting with great difficulty and showing considerable fibrosis. We have thus a condition of obstructive biliary cirrhosis in animals quite similar to that seen in man, and yet not caused by any experimental interference.

In *man* the question of the positive causation of cirrhosis of the liver by the obstruction to the flow of bile naturally has a different aspect, inasmuch as we are unable to produce experimentally this obstruction and to note the changes in the liver which would follow from it. We are, therefore, compelled in answering this question to examine the number of cases in which cirrhosis of the liver is seen in connection with a pathological obstruction to the ducts of the liver and to bring evidence in favor of the etiological relationship between the two by the number of cases of this combination which can be found.

Mangelsdorff, in 1882, reported 184 cases in which cirrhosis of the liver was associated with an obliteration of the bile-duct and in which there was no evidence that there were other etiological factors involved in causing this cirrhosis. Since 1882 we have been able to find twenty-four more cases in which as well an obliteration of the ducts of the liver seem to be the only causative agency in producing the chronic interstitial hepatitis. In all of these cases the usual factors of alcohol, syphilis, and poisonous intoxications which are seen in cirrhotic cases may be eliminated by careful study of the case-reports.

We thus have a total of 208 reported cases of cirrhosis of the liver in which the complete retention of the hepatic secretion and its subsequent action on the hepatic parenchyma is the only factor which can be shown to have any etiological influence in producing this cirrhosis.

This number at first sight seems rather small, especially that collection of cases since 1882; but if we remember, however, that Mangelsdorff's report includes all of the cases reported from 1822 to 1882, and that a very large amount of the work done on the liver occurred in the decade from 1870 to 1880, the apparent discrepancy between the

number of cases reported up to 1882 and since then is thus explained. Moreover, cirrhosis of the liver itself is comparatively rare, and to be able to quote a series of over 200 cases in which the factor of obliteration of the ducts alone is the only agency which can be cited as causing the cirrhosis is sufficient, from a pathological stand-point, to show that obliteration of the ducts and cirrhosis of the liver are definitely associated as cause and effect.

Etiology of this Cirrhosis seen in Obstruction. There are various views as to the actual cause of the cirrhosis of the liver which has been noted in connection with obstruction to the flow of bile. Observers have been divided in their opinions, and have maintained, on the one hand, that the stasis of bile alone was sufficient to cause cirrhosis, and, on the other hand, that there was always an inflammation of the bile-ducts, subsequent to this stasis, which caused a new growth of connective tissue in the liver. Charcot maintained that the retention of bile alone by its action on the liver cells was quite sufficient to cause the fibrosis seen in this organ; but Litten, on the contrary, considered that obstruction alone would not cause cirrhosis, but that there was in all cases an inflammation of the walls of the ducts. The latter cited in favor of his view a number of cases of gallstones where the stones were round and smooth and had caused no inflammation of the ducts and no cirrhosis of the liver. He contrasted these cases with those in which the gallstones were rough and sharp-edged, and had caused an inflammation of the ducts and the subsequent development of fibrous tissue. Litten, as well, operated on a number of animals under antiseptic precautions, ligating the ducts, but obtaining no cirrhosis; he did obtain a cirrhosis, however, by the injection of croton oil and other irritating substances into the common duct.

As we have pointed out above, the association of cirrhosis of the liver and the presence of rough, sharp-edged gallstones is explained by the complete obliteration of the ducts caused by the ulceration of these stones and the subsequent growth of scar-tissue quite as well as by the inflammation of the bile channels.

Gerhardt, in 1892, who had noted after ligature of the ducts the presence of many necrotic areas in the liver cells, endeavored experimentally to explain these necrotic areas in cases where there was obstruction to the flow of bile. He took one set of animals in which he ligated the common ducts, estimated the pressure of bile behind the ligature, and found it to be between 2 and 4 cm. of water. The livers of these animals showed necrotic areas. He now repeated the experiment in another set of animals, but drew off the bile and passed salt solution into the ducts under a pressure of 50 cm. of water. The livers of these animals showed no necrosis, and he therefore concluded that it was the long-continued action of the bile on the liver cell which

caused the slow cell death, not the pressure caused by the biliary stasis in the ducts themselves.

The *inflammatory origin* of the fibrous tissue seen in the livers of obstructive cirrhosis has been supported by a number of observations in which various micro-organisms were found in the liver. As we have stated above, in one of the cases in Montreal the number of these micro-organisms was so great as to lead to the belief that they might be a causal factor in producing the cirrhosis. Subsequent experiments, however, show that the majority of the livers of man and animals contain various forms of micro-organisms in their normal healthy condition, and, therefore, the mere finding of bacteria in cirrhotic livers cannot be considered as a proof that the cirrhosis is dependent upon the presence of these bacteria. In two at least of the cases studied here there was no evidence that bacteria were present to any greater degree than they are present in the normal hepatic parenchyma. Moreover, Vaughan Harley has recently sent to the Molson Pathological Laboratory in Montreal several specimens from the livers of the animals in which he had caused experimental cirrhosis. Dr. Adami has carefully studied these specimens in order to determine, if possible, whether micro-organisms are present or not, and he has uniformly failed to find any bacteria in these livers, either by the ordinary methods of staining, by Gram's method, or by the use of carbolthionin. It is natural to conclude, therefore, that from the experimental stand-point, at least, obstruction to the flow of bile will cause cirrhosis of the liver without infection of the bile channels.

In man, of course, this question has a somewhat different phase, for there is in practically all cases a considerable inflammation of the walls of the bile channels and often a slight inflammation of the smaller bloodvessels, the small-celled infiltration between the lobules often reaching an extreme grade, the appearance suggesting even the formation of small abscesses. Hence, it is impossible to find cases of cirrhosis of the liver in man associated with obstruction where one can say with certainty that the obstruction to the flow of bile has been the only factor concerned in causing the increase of fibrous tissue. We may thus conclude that the damming back of bile caused by the obliteration of the ducts does not always lead to the cirrhosis *per se*, but may lead to it as well by causing a certain amount of inflammation of the bile channels.

Even if we grant that the retention of bile and its poisonous action on the liver cells is the cause of the cirrhosis of the liver, we are still in some doubt as to the path which the bile traverses in order to come in contact with the hepatic parenchyma, for Vaughan Harley has recently shown that the jaundice which develops after the ligature of the bile-ducts will not supervene with the same rapidity if the thoracic duct be

tied as well. Evidently the lymphatic circulation is the path by which the bile reaches the different tissues and organs of the body, and it may be possible that the bile may reach the liver cells by passing from the bile-ducts through the lymphatic circulation and back again into the liver cells themselves.

Varieties of Biliary Cirrhosis. Under the title of biliary cirrhosis there have been described at least three different conditions—the biliary cirrhosis of children in India, Hanot's cirrhosis or hypertrophic cirrhosis with jaundice, and the cirrhosis dependent upon obstruction of the bile passages.

The first variety has been emphasized especially by Gibbons and McNally, who have reported a number of cases from the poorer classes of the inhabitants of East India. The children subject to this disease show during life a painless enlargement of the liver, a voracious appetite alternating with anorexia, a languid, fretful disposition, great thirst, fever, splenic enlargement, constipation, and clay-colored stools, with jaundice and eventually ascites, increasing up to the time of death. At autopsy the livers are enlarged, bile stained, typically cirrhotic, their microscopical examination showing, moreover, a mixed interlobular and intralobular type of cirrhosis, with a great degeneration of the liver cells and many increased bile-ducts. The cirrhosis of this organ is very irregular, often single groups of liver cells, or even single cells, being isolated and surrounded or replaced by connective tissue. It is thus really a pericellular cirrhosis. The etiology of this variety is supposed to lie in the use of unsuitable food and in the subjection to bad hygienic conditions, to which the children of the poorer classes in India are especially liable. It is not impossible that, like Hanot's cirrhosis, to which this variety seems to bear some resemblance, the cause of the disease of the liver may be primarily infectious.

Hanot's Cirrhosis. The cases usually recorded under the title of *biliary cirrhosis* correspond with fair accuracy to the description given by Hanot of his *hypertrophic cirrhosis with jaundice*, and Hanot himself makes use of the terms biliary cirrhosis in describing the disease which he has named. In this disease the main clinical symptoms are dull pain in the region of the liver; acute attacks of abdominal pain, resembling hepatic colic; a slight jaundice, increasing after each attack; a gradual increase in the size of the liver and in the circumference of the abdomen, with the maintenance of fair general health and an excellent appetite for some time. The stools are not clay-colored, but the urine contains an abundance of bile pigment; there is no ascites and no enlargement of the abdominal veins, although there may be intestinal hemorrhage; the fever, at first slight, may become marked, the temperature rising in the evening to 102° or 104° F., and the pyrexia assuming the character of hepatic intermittent fever. The bodily

strength is maintained, the emaciation is slow, and the course of the disease chronic.

There are, however, acute cases of Hanot's cirrhosis in which the patients die within a few months after their first observation by clinicians, having practically no jaundice and few biliary symptoms except an enlargement of the liver, but showing at autopsy a hypertrophic cirrhosis of the liver corresponding to the type described by Hanot, without obstruction to the flow of bile.

Pathologically, in Hanot's cirrhosis the liver is symmetrically enlarged and smooth; on section it cuts with difficulty, and has a jaundiced, dark-green appearance; the common and hepatic ducts are quite pervious and show no obstruction to the flow of bile, although the smaller ducts may be slightly dilated.

Microscopically, about the larger bile passages there is development of new fibrous tissue in concentric layers, this new tissue usually lying between the lobules, but at times penetrating within them, producing an intralobular as well as an interlobular type of cirrhosis. More frequently, however, the cirrhosis approaches the pericellular type—that is, there are individual groups of liver cells at the edges of the lobules, which are being cut off by the new connective tissue. There is a chronic diffuse angiocholitis and a periangiocholitis, but the new tissue is more of a replacement than of a productive inflammatory type.

We are in some doubt as to the etiology of Hanot's cirrhosis, and Hanot himself has not given any exact evidence to solve this problem. He has stated that the cirrhosis may be due to angiocholitis of the bile passages, to gallstones, and to obstruction to the flow of bile, and intimates that it may be infectious in origin. Goluboff, who has given the most accurate description of Hanot's cirrhosis published recently, attributes the fibrosis of the liver in his cases, which he described as biliary cirrhosis, to angiocholitis of the smaller bile-ducts, in which the retained bile causes a new growth of connective tissue between the lobules by the direct chemical or toxic action of the bile on the liver cells, the biliary stasis being due to the obstruction caused by the inflammation of the larger bile-ducts. The usual view, moreover, now maintained by the French school of pathologists is that Hanot's cirrhosis is caused primarily by infection, and is thus to be differentiated entirely from cases of cirrhosis of the liver seen in obstruction to the flow of bile caused by gallstones or by congenital obliteration of the bile-ducts.

Comparison of Different Varieties of Cirrhosis. Let us now compare Hanot's cirrhosis with those cases of obstruction cirrhosis which we have described, to see, if possible, whether the two conditions can be differentiated on pathological and clinical grounds.

In Hanot's cirrhosis the liver is uniformly enlarged, the surface smooth, without perihepatitis, the contraction to which all cirrhotic

livers are liable taking place in only a few isolated instances. It must be remembered, however, that there are cases in which patients pass through a cycle of symptoms characteristic of hypertrophic cirrhosis, with jaundice, and at autopsy reveal a small hobnailed liver; such cases are necessarily quite rare. The bile-ducts in this variety are seldom dilated, and when so only to a moderate extent, their lumen being always pervious.

In obstructive cirrhosis the liver may be enormously enlarged, as in the congenital cases, or it may approach in character the hobnailed liver of Laennec. There is usually considerable perihepatitis, the bile passages are always greatly dilated, and there is always, of course, a complete obstruction of some one of the ducts.

On section the livers in both diseases cut with great difficulty, revealing the presence of large amounts of new fibrous tissue distributed in islands between the hepatic lobules, the only difference between the two conditions here being the enormous dilatation of the bile-ducts which the obstructive cirrhosis reveals in contrast to the mild dilatation which Hanot's cirrhosis shows.

Microscopical examination reveals almost identical conditions in the livers of obstruction and the livers of ordinary hypertrophic cirrhosis with jaundice. In both cases the connective tissue develops about the bile passages, between the lobules of the liver, and may either lie between these lobules alone, causing an interlobular cirrhosis, or may penetrate within the lobules, causing an intralobular type. In both cases where the fibroblasts surround at the edge of the lobule individual liver cells the type of cirrhosis becomes more nearly that of a pericellular cirrhosis. There is the same inflammation of the smaller bile channels, the same congestion of the bloodvessels, and the same degeneration of the liver cells at the edge of the lobule. The reduplication of bile-ducts, as we have already shown, reaches an extreme grade in both cases. It is as yet impossible to say whether the wreath-like arrangement of these ducts which has been noted in cases of obstruction can be discovered as well in Hanot's cirrhosis, further study of the latter disease being necessary before any positive assertions can be made in regard to the appearance of this wreath-like distribution in hypertrophic cirrhosis with jaundice.

The autopsy findings in the two diseases differ only in the changes subsequent to the obstruction to the portal circulation which is seen in the cirrhosis of the liver dependent upon obstruction of the bile-passages—that is, œdema of the extremities, abdominal ascites, and a dilatation of the abdominal veins—a triad of symptoms which appears seldom or never in Hanot's cirrhosis.

From the *etiological* stand-point the two conditions naturally differ greatly in their causation: Hanot's cirrhosis is generally an infectious

process—that is to say, the presence of the symptoms and the pathological findings of Hanot's cirrhosis are better explained by the supposed entrance of micro-organisms into the biliary passages than they are by any other known factors. The changes seen in obstructive cirrhosis follow invariably the complete obliteration of some one of the bile-ducts, but both in Hanot's cirrhosis and in the variety of cirrhosis which we are especially considering we are compelled to look to the inflammation of the smaller bile passages as being the immediate cause of the production of the fibrous tissue between the lobules of the liver.

We thus see that from pathological grounds alone the changes seen in the two varieties of cirrhosis are quite similar, and that it would be impossible from our present knowledge to say, on examination of any particular variety of cirrhosis of the liver, whether we were dealing with simple hypertrophic cirrhosis with jaundice or with a case of obstructive biliary cirrhosis.

Clinically, the symptoms of the two diseases differ in the widest extent in their occurrence, in their intensity, and in the rapidity with which the patients succumb to the morbid changes. In hypertrophic cirrhosis with jaundice we have a slow, chronic disease, where the general health is maintained for some time; emaciation and wasting are slow, the appetite good, with a little gastro-intestinal disturbance. The jaundice is insidious in its onset, the increase of the icterus being coincident with paroxysmal attacks of hepatic colic. The urine is loaded with bile pigment, although the bile always finds its way into the intestines, as is shown by the presence of bile-stained stools. The liver is enlarged throughout the whole course of the disease except in rare instances, and when the contraction of this organ occurs a small amount of ascites may develop. Fever is a constant, and, in fact, a characteristic symptom. It is intermittent in type and forms the basis for that pyrexia now constantly described as hepatic intermittent fever. The patient usually after a long period, during which he alternates between a fairly good state of health and attacks of colic, fever, and jaundice, very gradually loses his flesh and strength, and only at a late date succumbs to the disease process.

In obstructive cirrhosis, however, the clinical picture is absolutely different: the disease is acute in its onset and in its course: emaciation and loss of strength are rapid, anorexia and vomiting are extreme, the headache severe, the stools invariably clay-colored, and the portal circulation greatly impeded, leading to the development of ascites and the œdema aforementioned. Fever does not occur in uncomplicated cases of obstructive cirrhosis; the jaundice, sudden in its onset, is extreme from its first appearance, a diminution of the yellowish color of the skin and conjunctiva practically never occurring. The systemic disturbances are marked, especially the itching of the skin, with hemor-

rhages beneath its upper layer, and the headache and vomiting. The disease is rapidly progressive, the patients have no intermission of their characteristic symptoms, but rapidly lose strength and flesh, sinking into that condition of coma or of auto-intoxication which is only ended by death itself.

CONCLUSIONS. From the study of the cases of cirrhosis seen in Montreal, and from a review of the cases reported by other authors, together with the observations of experimental pathologists, we are justified in drawing a number of general conclusions:

Complete obstruction to the flow of bile in *animals* produces a cirrhosis of the liver, interlobular in character, of considerable extent, provided the animals are allowed to live a sufficient time after primary operation. This cirrhosis is due to the damming back of bile *per se*, and not to the inflammation of the walls of the smaller bile channels.

In *man*, complete obliteration of the bile-ducts caused by pathological changes leads to a cirrhosis of the liver which cannot be explained simply by the damming back of bile, but must be considered to have, as an added etiological factor, that inflammation of the walls of the biliary channels which is invariably present.

The type of the cirrhosis of the liver seen in man subsequent to obstruction of the bile passages cannot be differentiated anatomically from the type of liver seen in Hanot's hypertrophic cirrhosis with jaundice.

The symptom-complex of obliterative cirrhosis is so absolutely different from Hanot's cirrhosis in the variety of its symptoms and in the frequency with which they occur as to justify the statement that *obstructive biliary cirrhosis* is a distinct morbid condition quite apart from any other variety of cirrhosis of the liver.

SYMPTOM-COMPLEX OF HANOT'S CIRRHOSIS AND OBSTRUCTIVE BILIARY CIRRHOSIS.

Symptoms.	Hanot's Cirrhosis.	Obstructive Cirrhosis.
Course of disease	Chronic.	Acute.
General health	Good.	Poor.
Emaciation	Slow.	Rapid.
Loss of weight	Slow.	Rapid.
Intermission of symptoms	Common.	Does not occur.
Fever	Common.	Rare.
Anorexia	Rare.	Common.
Good appetite	Common.	Rare.
Vomiting	Rare.	Common.
Jaundice	Slight at first, increasing.	Deep from the first.
Clay-colored stools	Rare.	Constant.
Bile-stained urine	Common.	Constant.
Enlargement of liver	Common.	Common.
Contraction of liver	Rare.	Common.
Ascites	Rare.	Common.
Œdema of extremities	Rare.	Common.
Caput medusæ	Rare.	Common.

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DORSAL DISLOCATION OF THE TRAPEZOID.

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DORSAL dislocation of the trapezoid, unaccompanied with fracture or dislocation of other bones, occurs so rarely that it may be looked upon as a surgical curiosity. I could find in the *Index Medicus* and in the *Index Catalogue of the Library of the Surgeon-General's Office* the report of only one case. This was recorded by Geo. W. Gay in the *Boston Medical and Surgical Journal* in 1869.

In this paper I shall give a summary of Gay's case, and shall report a case of injury that came to me and was diagnosed and treated as a case of simple uncomplicated dorsal dislocation of the trapezoid.

Dr. Gay's patient injured his wrist by striking a post in such a

manner that the force was brought to bear on the distal ends of the metacarpal bones of the thumb and index finger. At the time the injury was produced the wrist was in the straight position and the forearm in extension. On examination sixteen hours after the accident occurred there was found an irregular quadrangular swelling situated proximal to the base of the metacarpal bone of the index finger. The swelling seemed to extend about one-quarter of an inch beyond the level of the dorsal surfaces of the other carpal bones. It was not very tender, was of bony hardness, and could not be reduced by pressure. The metacarpal bone of the index finger did not seem shorter than normal. No crepitus was obtained, and no other injury to the wrist or hand observed.

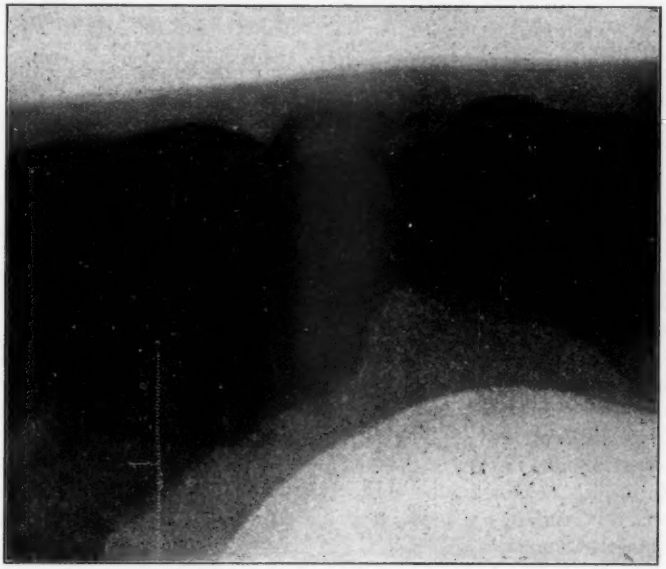
Dr. Gay was unable to reduce the deformity. He extended the hand on the forearm at an angle of forty-five degrees and held it in this position with an anterior splint and with adhesive plaster. At the end of two weeks this dressing was removed. Five weeks later—that is, seven weeks after the injury occurred—the patient was able to move the injured wrist freely and was able to use it without causing him pain. But the deformity remained the same as it was at the time that Dr. Gay first saw the case.

The history of the case that I desire to report is as follows:

A policeman, aged thirty-nine years, struck a man with his fist in such a manner that the entire force of the blow was brought to bear on the distal end of the metacarpal bone of the index finger. The wrist-joint and elbow-joint were slightly flexed at the time the blow was delivered. Immediately afterward he suffered from pain in the injured part. This was not very severe, but was increased by movement of the hand or wrist. The pain gradually diminished till I saw him, two hours after the injury. At this time he was entered as a patient in Cook County Hospital, Chicago. On examination I found the right wrist slightly flexed. Just proximal to the base of the metacarpal bone of the index finger was a cuboidal swelling, of bony hardness, that extended nearly a centimetre beyond the dorsal surfaces of the other carpal bones. This swelling was not easily movable, was not painful, and was but slightly tender on pressure. No crepitus was obtained. The soft structures showed very little swelling; there was no evidence of extravasation of blood. No shortening of the metacarpal bone of the index finger was found, and no widening of the wrist could be made out. No attempt was made to reduce the deformity at this time. Six hours later the fluoroscope was used and showed the injury to be a simple dorsal displacement of the trapezoid.

Eight hours later I attempted to reduce the deformity. In this I was only partially successful. I made extension on the index finger, and, at the same time, made direct pressure on the dorsal swelling. The deformity was partially reduced, but the displaced bone still projected nearly one-fourth of an inch beyond the dorsal surfaces of the other carpal bones. I then held the hand in a straight position—neither flexed nor extended on the forearm—and wound a piece of

adhesive plaster, two inches wide, several times around the wrist just above the carpal extremities of the metacarpal bones of the thumb and little finger. The extremity was placed in a sling, and the patient was told to return to the hospital on the third day. He did not come back, and I did not succeed in finding him till fifteen weeks later. At this time the deformity was the same as it was after I had attempted to reduce it, except that the bone was firmly fixed and its borders and angles could not be made out. There was no limitation of motion in the wrist-joint.



Skiagraph showing dislocated trapezoid.

The patient told me that he carried the injured member in a sling for five days. He then went to work on the police force, and on the tenth day after the injury he removed the adhesive plaster. He said that the wrist had pained him somewhat when he used it, but that it was gradually improving and was nearly as strong and as useful as it ever had been.

I tried experimentally, on the cadaver, to produce dorsal dislocations of the trapezoid by simulating the mode of production in these two cases. I had the arm and wrist held both in the flexed and in the straight positions, and applied force to the distal end of the metacarpal bone of the index finger and to the metacarpal bones of the index finger and thumb. In each of the twelve wrists that I experimented on either the metacarpal bone of the index finger was fractured, or the ligaments

were so torn as to render it impossible to apply force in the direction desired. In no case was I able to produce a simple uncomplicated dorsal displacement of the trapezoid. From the results of the foregoing experiments it seemed probable to me that the exciting cause—trauma—is not, under ordinary circumstances, sufficient to produce the injury being discussed. I therefore looked for predisposing factors, and my attention first turned to the anatomy of the parts involved.

The trapezoid is wedge-shaped, the base of the wedge forming the dorsal surface of the bone. Its superior and inferior surfaces are concave from before backward, but the concavity, in most cases, is slight, and doubtless aids but little in keeping the bone in its normal position. Therefore, pressure brought to bear on the lateral or superior or inferior surfaces of the trapezoid will tend to displace it in a posterior or dorsal direction. Under ordinary circumstances, it is probable that pressure on the lateral surfaces of the trapezoid has very little effect in displacing it dorsally; and force applied to the distal end of the metacarpal bone of the index finger is transmitted to the trapezium and os magnum. These observations led me to believe that the shape of the trapezoid predisposes it to dorsal displacement, and this I verified by the following experiments: After freeing the trapezoid from the other carpal bones, and after separating the trapezium from the os magnum so that force applied to the distal end of the metacarpal bone of the index finger would affect the trapezoid, I was able, on the cadaver, to produce simple dorsal dislocations of this bone by holding the forearm and wrist firmly and applying force to the distal end of the second metacarpal. Slight flexion of the metacarpi on the carpal bones facilitated the displacements. In the normal adult this dislocation is prevented from occurring by the presence of the soft tissues—principally by the ligaments.

The ligaments of the trapezoid are usually divided, for description, into dorsal, palmar, and interosseous. The palmar ligaments are stronger than are the dorsal, and the dorsal ligaments of the trapezoid are less strong than are the corresponding ones of any other carpal bone of the second row except the trapezium. The interosseous ligament between the trapezoid and the os magnum is situated near the dorsal surfaces of the bones. Morris says that there is no interosseous ligament between the trapezoid and the trapezium. Other writers claim that this ligament is present in the majority of cases. I was unable to find a report of examinations made to determine the percentage of cases in which this ligament is absent. I have examined fourteen cases, and have found in each case an interosseous ligament between the trapezoid and the trapezium.

It occurred to me that I might be able to produce an artificial dislocation of the trapezoid in cases in which the interosseous ligament

between this bone and the trapezium was absent or had been divided. I therefore cut the ligament and then tried to dislocate the trapezoid dorsally by holding the forearm and wrist firmly and applying force to the distal end of the metacarpal bone of the index finger. I did this in ten cases, but failed to produce a single uncomplicated dorsal displacement. I then, in the same cases, divided the dorsal ligaments between the trapezoid and the metacarpal bone of the index finger. After doing this I was able, by using the method described in the foregoing, to dislocate the trapezoid posteriorly in every case. But the results were not all alike—sometimes the bone would be displaced a distance corresponding to one-fourth or one-half of its anterior-posterior diameter; again, it would be thrown dorsally only a few lines.

These observations and experiments led me to believe that it was probable that in Gay's case, and in the case that came under my care, there was a congenital weakness or absence of the ligaments, or a maldevelopment of the carpal bones, or a combination of these conditions which rendered it possible for an uncomplicated dorsal dislocation of the trapezoid to be produced.

REVIEWS.

THE PRINCIPLES AND PRACTICE OF HYDROTHERAPY: A GUIDE TO THE APPLICATION OF WATER IN DISEASE. FOR STUDENTS AND PRACTITIONERS OF MEDICINE. BY SIMON BARUCH, M.D., Visiting Physician to the J. Hood Wright Memorial (formerly Manhattan General) Hospital; Consulting Physician to the Montefiore Home for Chronic Invalids; Member of the New York Academy of Medicine; formerly Gynecologist to the Northeastern Dispensary; Physician for Eye, Ear, and Throat at the Northwestern Dispensary of New York City; Physician and Surgeon to the New York Juvenile Asylum, and Chief of the Medical Staff of the Montefiore Home for Chronic Invalids. With numerous illustrations. 8 vo., pp. xx., 504. New York: William Wood & Co., 1899.

THE preface to this excellent manual upon an important and much neglected therapeutic art states that, different from all other works on its subject, "this book is written by a general practitioner for the guidance of his colleagues." It represents the observations of the author, who has labored in every branch of medicine, and has gathered the data upon which his recommendations are based, from a private and hospital practice of more than thirty years; including a special institution practice of seven years, in which over one hundred thousand "hydriatric treatments" have been recorded. The author begins by defining hydrotherapy as "the method of applying water in disease." We should substitute for "disease" in this definition—the management of the sick. The principle, however, is the same. Hydrotherapeutics is not concerned with cold water alone, nor with external application only, but includes the therapeutic use of water in any form, from ice to steam, internally and externally. Nor is hydrotherapy to be confounded, as the author justly remarks, with "hydropathy;" the latter barbarous term being applied to the quack systems of the treatment of all diseases by means of the external use of water, and, principally, cold water, following Priessnitz. Other terms referring to the subject in general are the adjectives "hydriatric" and "hydriatic." The former is a short and expressive substitute for the adjective "hydrotherapeutic;" the latter an adjective in common use qualifying nouns connected with the use of water in medicine or for other purposes. Its correct derivation the author has been unable to obtain, and he would like to have it banished from medical terminology. The book is divided into two parts.

Part I. deals with principles. It includes studies of the physical properties of water and of its effect upon the human body in health, the various subdivisions of this part making four chapters of eighty pages. Diagrams, charts, and tables are given to illustrate important data. The anatomy and physiology of the skin necessarily receive consider-

able attention, as the results of external applications of water are largely due to the response of the nerves and vessels of the skin to their therapeutic environment. The skin, by which the author means not merely the cutaneous structure, *per se*, but also the vascular, nervous, and glandular structures bound up therewith, has important functions as an organ of sense, general and special, as an organ of secretion and excretion, as a supplementary organ of respiration, and as a heat regulator; the latter being an important property in connection with hydrotherapeutics. Temperature effects, however, are not the only results of the external application of water, but mechanical, and to a minor degree chemical, effects are produced as well. These act as stimuli to the peripheral sensory nerves, irritation being conveyed to portions of the central nervous system, and thence reflected by the motor fibres to the various parts that the physician desires to influence; or changes of the local innervation of the part which receives the applications may be produced by effects upon the ganglionic centres in the course of the nerve supply of the vessels. In addition, there are direct effects upon the vessel walls, upon the secretory structures, and, mechanically, upon the superficial or even deep structures as a whole, according to the susceptibility of the part or of the organism, and the force and suddenness of the impact. The author considers, therefore, the influence of water applications upon circulation, respiration, temperature, tissue change, and secretion, setting forth the results of experimenters with fulness and some critical comment. Among the original observations reported in this connection are studies of the effect of various hydriatric procedures upon the blood constituents in health and disease, made at the Montefiore Home.

Part II., consisting of 23 chapters and 350 pages, refers to the practice of hydrotherapy. The author has made no attempt to be encyclopedic in description, citation, or history, but confines himself to such procedures as personal experience has proved to be useful. Each procedure is described in detail, and illustrated by drawings. Its rationale is then fully set forth, and, finally, its therapeutic indications enumerated. In the discussion of certain subjects illustrative cases are cited or statistics referred to, this latter feature being specially notable in the section upon the Brand treatment of typhoid fever. The author's well-known insistence upon precision in the technique of hydrotherapy is admirably illustrated in this portion of his work. As he has somewhere remarked, to tell a patient to "use cold water" or "use hot water," is like telling a patient to take "some quinine" or "some morphine." Indeed, the latter is a little nearer accuracy, and no more dangerous; it at least indicates the method of administration. Hydrotherapeutic procedures must be carefully adapted to the individual patient and the particular stage of general or special morbid processes present at the time of the application. The temperature, duration, pressure, and manner of application, to the minutest detail, must be specifically prescribed. Only thus can hydrotherapeutics be scientifically practised and useful results anticipated.

The author describes, under the head of external applications, the following procedures: Ablution, the half bath, affusion, the sheet bath, the cold rub, the wet pack, the wet compress, the full bath (the latter subject being divided into the following heads: the cold full bath, the warm full bath, and the hammock bath), the douche, the hip

bath, and irrigation (including lavage, enteroclysis, irrigation by the genito-urinary canals, and miscellaneous forms); then come methods of cooling and heating internal parts, including the prostatic cooler, the perineal douche, the rectal cooler, the cooling sound used in prostatitis, etc.; and a chapter is devoted to the internal uses of water. Several chapters are devoted to the practical application of hydrotherapy in acute and chronic diseases; the various therapeutic indications scattered through the book under various procedures being summarized and supplemented under the headings of special diseases.

An excellent chapter on "the hydriatric prescription" gives wise directions as to the method of ordering applications, and warns against the errors commonly committed. The concluding chapter is devoted to an historical epitome, and a few earnest words as to the necessity for instruction in hydrotherapy. The author is entitled to much credit, not alone for his earnestness in introducing important therapeutic measures into general use in this country (for, although he had been preceded by many in urging these measures, he was the first to systematically devote himself to the subject, and it is he who does, not he who thinks, that deserves the credit in such matters), but also for the clear and complete manner in which he has treated the subject in his book. There is no longer an excuse for anyone remaining uninformed on the subject of hydrotherapy. The knowledge is no longer far off in the heaven of French books or the repulsive depths of German books, but here at home before us, this day, in our own tongue, to learn and to do. Dr. Baruch's book contains all the essentials and many details and valuable suggestions from personal experience. Perhaps critical pruning and condensation, and some rearrangement of material to make discussions more consecutive, may be improvements in future editions.

One naturally turns with great interest to the discussion of the Brand treatment of typhoid fever, with which Dr. Baruch's name is historically and indissolubly connected, and he finds a full and careful discussion, with, perhaps, too much citation of individual opinions and too much insistence upon the strict execution of Brand's method. The author dismisses most of the objections urged against the latter as puerile or absurd. It is true that some of the objections which he cites may thus be characterized, but not all the objections made. The reviewer's practice is to bathe typhoid fever patients when they need it, at such a temperature of patient and bath, for such a time, and in such a manner, as the particular indications of the particular case and particular time commend. He has had no cause to regret this method, carried out for more than fifteen years, and never having been led astray by the antipyrine seduction, lacks perhaps the enthusiasm of the convert which Dr. Baruch here exhibits. There is no doubt that the routine application of Brand's systematic treatment to every case of typhoid fever exhibiting a temperature of 103° F. in the rectum, is better and will save more lives than any other routine treatment; but we consider all routine treatment to be bad treatment. A general plan for the circumstances presented in a specific group of morbid and reactionary (salutary) phenomena of known succession and coincidence, to be modified in accordance with the variations of succession, coincidence, and degree of these phenomena, and the appearance of irregular phenomena, is always better. Thus the Brand bath may be taken as the type of procedures for the typical case of typhoid fever; but as we so rarely see

the typical case of typhoid fever in actual practice, so is there rarely occasion continuously to apply the typical Brand bath with systematic rigor. Hydrotherapeutic measures by all means, and bathing in the majority of cases, and the full bath at a temperature below 80° F., usually, and at 65° F. not rarely; but individual modification, and modification from day to day, perhaps modification from bath to bath, are always necessary. To substitute for the chart of continuous pyrexia with moderate and regular remissions and exacerbations characterizing typhoid fever, a temperature-chart of violent fluctuations resembling hectic fever, is not desirable; and we must again record our objection to any procedure which in a single bath reduces the temperature more than about 2° F. With Dr. Baruch we look upon the effects of cold bathing as affecting nutrition in general, and neuro-vascular processes specially, rather than thermic processes merely, temperature simply being an indication of the activity of morbid and reactionary phenomena; but we prefer these to be altered gradually rather than suddenly, especially when the sudden change is followed by equally sudden return to previous force.

Other important articles are those on the uses of water in cases of acute lobar pneumonia, bronchopneumonia, phthisis, neurasthenia, dyspepsia, and cholera. While our own experience is not always on all-fours with the author's, his acquaintance with the methods discussed and their special applications is so much the greater, that we cannot refuse to give his views correspondingly greater weight.

The profound impressions which hydriatric procedures are capable of making upon the functions of organic life cannot be studied without deepening the conviction that these effects ought to be utilized in medicine to a far greater extent than is now the case. It is impossible for the general practitioner to become thoroughly acquainted with their technique or to take time to apply them in all suitable cases, but he should know their rationale and their scope of usefulness. Whenever possible, and notably in the acute infections, he should apply them with the help of trained nurses or of members of the family; and for chronic affections requiring special facilities, knowledge, and apparatus, there should be in every important medical centre an institution supported by the profession at large, to which patients could be referred with general or special prescription. At present, patients who could be benefited by the rational and scientific use of water are deprived of the treatment, or dependent upon the enthusiasm and persistence of individual physicians; and there are not many Baruchs. S. S. C.

A MANUAL OF OPERATIVE SURGERY. By L. A. STIMSON, B.A., M.D., and JOHN ROGERS, B.A., M.D. Philadelphia and New York: Lea Brothers & Co., 1900.

AN operative surgery to be of the greatest usefulness should be concise and clear in statement and be well illustrated. Stimson has in this edition succeeded in meeting these requirements in the first instance. The illustrations are good, but there is still lacking something to make them entirely satisfactory. The descriptions of operative procedures are

very satisfactory. I know of no manual that is so well adapted to the needs of the student and practitioner. Anatomical plates are wisely used to render intelligible various steps. The transverse sections of the extremities in the part devoted to the ligature of arteries at different levels are helpful. Operations upon the intestines are well illustrated; many special operations are described. A very attractive method has been used throughout the greater part of the *Manual* in that the procedures of different surgeons are alluded to, and valuable references are made to articles in medical literature where complete descriptions of original methods may be found. The *Manual* is worthy to occupy a place beside such classical books as Treves' *Operative Surgery*.

C. L. S.

Atlas and Epitome of Special Pathologic Histology. By Docent Dr. Hermann Dürck. Edited by Ludvig Hektoen, M.D. Philadelphia: W. B. Saunders & Co.

As in the other books of this series, the illustrations are the special feature. The subject of pathological histology is perhaps more susceptible to this form of exploitation than any other in the domain of medical science, chiefly because of the excessive clearness and fine definition of the preparations ordinarily studied and the employment of staining methods that not only accentuate this clearness, but add a not wholly inartistic element to it.

Criticising, then, the illustrations, we can say without hesitation that they are perfect from their stand-point. Evidently preparations have been carefully made, then copied in water-colors, and the lithographs prepared from these. The details are accurately given, the colors are clear and approximate so closely to those obtained by the ordinary staining methods that they may, for all ordinary purposes, be considered identical. There has always been some doubt on the part of the makers of pathological histological text-books as to whether photo-micrographs are to be preferred to free-hand drawings; of course, it has been recognized that there is much to be said on both sides. The photograph is more accurate, but on account of the ability of the observer to change the focus continually and to study the relations of parts lying in different planes, it is doubtful if the drawing does not reproduce the object seen with greater fidelity. We are certain, at any rate, that the student can profit more by plates such as are given here than by the more elaborate photo-micrographs. The expert, however, with a fairly good collection of specimens in his laboratory, would prefer the latter. The preparations here reproduced represent the commoner morbid alterations of tissues; but as this book is prepared especially for students, a more admirable selection of subjects could with difficulty have been made.

Criticising the selection of plates and the methods of staining employed, we can find little that is not praiseworthy. We wish to call particular attention to the thoroughness with which the modern methods have been depicted. There are several plates in which the results of staining by Sudan III. have been employed. In the section on diseases of the bloodvessels, pictures of the elastica stain have been repro-

duced. Weigert's fibrin method, of course, is familiar, and is part of ordinary laboratory technique; nevertheless, some of the illustrations in which it has been employed are exceedingly beautiful. It might, perhaps, have been advantageous to have introduced a few plates showing the specific reactions of certain degeneration products, such as the amyloid substance with gentian violet, or mucin with thionin. Von Gieson's stain does not appear to have been employed as frequently as the beauty of its results would warrant.

As for the text, but little can be said; it is clear and concise, and presents the present status of the subject about as well as it could be presented within these limits. Professor Hektoen has gone over it carefully, and his additions, while not nearly as numerous as we could have desired from him, are all of considerable value. J. S.

A Practical Treatise on Diseases of the Skin, for the Use of Students and Practitioners. By James Nevins Hyde, A.M., M.D., Professor of Skin, Genito-urinary, and Venereal Diseases, Rush Medical College, Chicago; Dermatologist to the Presbyterian, Augustana, and Michael Reese Hospitals of Chicago; and Consulting Dermatologist to the Chicago Hospital for Women and Children; and Frank Hugh Montgomery, M.D., Assistant Professor of Skin, Genito-urinary, and Venereal Diseases, Rush Medical College, Chicago; Professor of Skin and Venereal Diseases, Chicago Clinical School; Attending Physician for Skin and Venereal Diseases, St. Elizabeth Hospital, Chicago. Fifth revised and enlarged edition, illustrated with 3 engravings and 24 plates in colors and monochrome. Philadelphia and New York: Lea Brothers & Co., 1900.

The esteem in which this standard treatise is held by the profession is amply attested by the fact that another edition has been found necessary two years after the appearance of the fourth. Although the changes are comparatively unimportant, owing to the brief period which has elapsed since the fourth edition was issued, the revision has been done in no perfunctory fashion, alterations and additions being plentifully scattered throughout the book, bringing it fully up to date. Two new chapters have been added—one on Porokeratosis, that very curious affection to which Mibelli first called attention, and one on Blastomycetic Dermatitis—a subject of great and apparently growing importance, both from a clinical and pathological stand-point, and one upon which the authors speak with authority. The value of the volume has been materially enhanced by the addition of a number of full-page illustrations, which are excellent examples of the fidelity with which photography may reproduce the appearances of cutaneous eruptions in properly selected cases. Such illustrations really illustrate, and are of the greatest value in helping to make plainer the descriptions of disease found in the text.

We note with especial satisfaction the wise conservatism of the authors in discussing the etiology of eczema. Under the influence of the teachings and writings of that indefatigable worker, Unna, and his

followers, there is a great and constantly growing tendency among recent writers to place this affection among the maladies due to micro-organisms; but we quite agree with our authors that the etiological relationship of the micro-organisms found in eczema is by no means yet determined, and that the causes of this protean affection are many and varied.

In conclusion, we can heartily commend this treatise as one of the best in the language, and a reliable guide for the student and practitioner.

M. B. H.

INJURIES TO THE EYE IN THEIR MEDICO-LEGAL ASPECT. By S. BAUDRY, M.D. Translated from the original by ALFRED JAMES OSTHEIMER, JR., M.D., of Philadelphia, Pa. Revised and edited by CHARLES A. OLIVER, A.M., M.D. With an Adaptation of the Medico-legal Chapter to the Courts of the United States of America, by CHARLES SINKLER, ESQ. Philadelphia, New York, and Chicago: The F. A. Davis Co., 1900.

As stated in the preface, this monograph was written with the idea of guiding the legal expert in trying cases where a profound knowledge of both the internal and external diseases of the visual apparatus is demanded, as well as to aid the ophthalmologist in estimating accurately the damage to the individual caused by injury. It contains a concise account of the traumatic lesions of the eye and its adnexa, especially from a prognostic stand-point.

The book is very readable, and the translation, which the reviewer has had an opportunity of verifying, has been conscientiously performed. The editor has fulfilled his duties with his usual care, and the work deserves to be well received by the profession. W. C. P.

NERVOUS AND MENTAL DISEASES: A MANUAL FOR STUDENTS AND PRACTITIONERS. By CHARLES S. POTTS, M.D. Philadelphia and New York: Lea Brothers & Co., 1900.

IN this small work the author has given to the student a clear, concise, and easily comprehended presentation of the subject of nervous and mental diseases. The different subjects are very thoroughly treated for so small a work. The treatment of many of the symptomatic diseases under separate headings adds to the value of the book. The neuron theory is accepted and made use of in describing the system diseases of the spinal cord. This, however, is of questionable value in a book of this size, inasmuch as it lends an element of uncertainty to many statements of little practical significance to the student. Wood's classification of the insanities is given, this section being up to the standard of the rest of the work. The book is well written, profusely illustrated, thoroughly indexed, and printed on an excellent grade of paper. The busy practitioner or student whose limited time precludes the use of more extensive treatises will find in this one of the best of the shorter works on this subject. D. J. McC.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Etiology of Rheumatic Fever.—POYNTON and PAINE (*The Lancet*, September 22, 1900, p. 861, and September 29, 1900, p. 932) have isolated from eight successive cases of rheumatic fever a diplococcus which they believe to be a cause of rheumatic fever. They do not claim that the organism is constantly present in rheumatism, nor do they definitely assert that it is the only cause of the disease.

They first give a concise review of the literature on the bacteriology of rheumatic fever, including among others the important contributions of Chvostek, Singer, Achalme, Thiroloix, Griboulot, Westphal, and Wassermann. Granting that rheumatism is a microbic disease, the researches of the various investigators present a diversity of views.

The first view is that there is no specific organism, but that rheumatism is but a form of septicæmia which owes its origin to staphylococcal and streptococcal infection.

According to the second view a specific diplococcus is regarded as the cause of rheumatism. This is the view to which the authors adhere with some limitation to the term "specific."

The third view maintains that the cause is a specific bacillus. Achalme and his followers were supporters of this view.

The fourth view raises the question of a mixed infection of bacilli and cocci, an analogy to which is supplied by diphtheria.

According to the fifth view, rheumatic fever is not a disease sui generis, but a particular reaction of the tissues to varied infections.

Poynton and Paine commenced their investigation with the intention of confirming, if possible, Achalme's results. They were unable to obtain a

bacillus morphologically resembling the anthrax bacillus either in the cultures or in the tissues as Achalme had done. On the contrary, they obtained in eight successive cases a diplococcus which grew in liquid media in streptococcal chains. This organism is in the form of minute cocci associated in pairs, the individual elements of which vary somewhat in size but average 0.5 μ in diameter. In liquid media they grow in chains of varying length; in solid media in masses that resemble the arrangement of staphylococci. The organism grows both aërobically and anaërobically, but better in the latter way. It grows best in a medium of milk and bouillon slightly acidified with lactic acid. The cocci stain with the various aniline dyes, but are easily decolorized by Gram's method.

This organism was isolated in eight successive cases of acute rheumatism. It was present in pure culture in five cases. It was obtained (*a*) from the blood of living patients suffering from acute rheumatic pericarditis; (*b*) from the pericardial fluid and from the fragments of granulations removed from the valves after death, and (*c*) from the throat of living patients suffering from rheumatic pericarditis. The organism was isolated in pure culture from the joint exudation, heart-blood, urine from the bladder, and cerebro-spinal fluid of rabbits that had been inoculated with a sufficient dosage. They have demonstrated the diplococcus in the cardiac valves, pericardium, and tonsils, and in a nodule in fatal cases of rheumatism. They also demonstrated it in the cardiac valves, pericardium, joint exudation, kidneys, liver, connective tissues, pleuræ, cerebro-spinal fluid, lungs, and urine of rabbits inoculated intravenously.

The changes in the rabbit following the intravenous injection of this organism are very suggestive of its close relationship with the etiology of rheumatic fever. It produces a polyarthritis, bursitis, and tenosynovitis. The polyarthritis may completely disappear. Where the arthritis persists the joint effusion becomes turbid with fibrin and cellular elements; in other cases the joint effusion is clear. It causes a multiple valvulitis and pericarditis, both non-suppurative. It produces plastic pleurisy and pneumonia and a fatty degeneration and destruction of the muscle fibres of the myocardium similar to that found in the human heart in severe rheumatic carditis. It has not led to suppurative foci in the viscera. The urine is acid and loaded with urates. The clinical symptoms are characterized by multiple painful joint swellings, wasting, and moderate pyrexia.

Poynton and Paine, in discussing the point whether this organism is the cause of rheumatic fever, say that, in the face of the researches of several competent observers, they cannot claim that this diplococcus is the only cause of rheumatic fever. That it is one cause they think they have proved to all practical purposes by their investigations. They further believe it highly probable that it will prove to be the cause of all cases of rheumatic fever which conform to the usual type of the disease, for the disease in man, as they state, is a very definite one, and therefore probably caused by a specific micro-organism.

Some Points about Post-diphtheritic Paralysis.—MEYER (*The Lancet*, September 22, 1900, p. 869) made an analysis of the cases of diphtheria admitted to the Park Hospital, London, in 1899, in which paralysis or paresis

developed as a sequel. There were 1316 cases of diphtheria admitted to the wards. Of these, 275 developed post-diphtheritic paralysis or paresis; in other words, about one-fifth of the cases. There were 147 cases in males and 128 in females. As the numbers of males and females admitted were about equal, the records show that paralysis was commoner in the former. Of those who suffered from paralysis there were 80 deaths (29 per cent.), 44 occurring in males (55 per cent.) and 36 in females (45 per cent.).

Cardiac paralysis was the cause of death in the largest percentage of cases. It caused death in 64 of the 80 fatal cases, or in 80 per cent. The great proportion of cases in which cardiac paralysis developed was in children between two and nine years, the majority being in the sixth to seventh year. After nine years of age there was a great falling off in these cases. The average date on which symptoms of cardiac paralysis appeared after the onset of the disease was the seventh day, the extremes being the first and thirty-sixth day. The average duration of life after symptoms of cardiac paralysis were first noted was the seventh day.

Diaphragmatic paralysis or paresis was present in 21, or 7.6 per cent., of the 275 cases. Of these, 11 cases terminated fatally. With only one or two exceptions the diaphragmatic paralysis was associated with paralysis of some other muscles.

There were 110 cases where the palate was alone affected, or 40 per cent. of the total number of cases. Counting the cases in which it was affected alone or in association with other muscles there were in all 166 cases, or 60 per cent., of the total.

Other muscles less frequently involved but in which the percentages are given were the ciliary, internal and external recti, facial, laryngeal, and muscles of deglutition.

It is to be regretted that no reference is made to the form of treatment of diphtheria carried out, and no statement is made concerning the effect that antitoxin had on the subsequent development of paralysis.

Experimental Proof of the Mosquito-malaria Theory.—That malaria is transmitted from man to man by a particular species of mosquito is now accepted by all biologists and medical men who have given proper attention to the matter. Considerable skepticism is still found, however, in the ranks of the medical profession and among the general public. In order to convince these skeptics of the importance of the mosquito in transmitting malaria, MANSON was instrumental in having carried out, during the past summer, two most convincing experiments.

Grassi and Bignami had proven that the mosquito could transmit malaria from man to man. As their experiments were carried out in Rome, which is situated in the midst of a highly malarial district, there might be some ground for the objections of the skeptics. Manson's son, Mr. P. Thurburn Manson, of Guy's Hospital, volunteered to be the subject of the first experiment. He is twenty-three years of age, was born in China, and had gone to England at the age of three. Since then he had never been abroad and had never been in any district in England reputed to be malarious. Arrangements were made with Bignami and Bastianelli for relays of mosquitoes (anopheles) to be forwarded from Rome as soon as possible after they

had fed on patients suffering from benign tertian malaria. The first consignment arrived at the London School of Tropical Medicine on July 5th. Possibly one of these bit the subject of the experiment. Mosquitoes of a second consignment he allowed to bite him on August 29th, August 31st, September 2d, and September 4th. Of a third consignment, twenty-five bit him on September 10th and ten on September 12th. Manson remained perfectly well until September 13th, when headache, fever, general lassitude, and aching pains ensued. Fever continued on the 14th. On the 15th he felt chilly and his temperature rose to 103.6° F. On the 16th and 17th there were exacerbations of fever. The examination of the fresh blood on the latter two days showed typical tertian malarial parasites. Their presence was confirmed by several competent observers. Quinine was started on the 17th, and the fever and parasites promptly disappeared. The convincing nature of this experiment seems to require no comment.

In the second experiment a hut, constructed in London, was shipped to Italy and erected in the Roman Campagna at a spot where Dr. L. Sambon, who had gone to Italy in connection with this experiment, found that the permanent inhabitants all suffer from malarial cachexia, and where the field laborers who come from healthy parts of Italy to reap the crops after a time all contract malaria. Dr. Sambon, Dr. Low, Signor Terzi, and their two Italian servants entered on residence in the hut early in July. The only protection against mosquito bites and fever employed was mosquito-netting, wire screens on the doors and windows, and mosquito nets around their beds. During the day they went about the country quite freely. They were always careful to be indoors from sunset to sunrise. Up to September 21st, the date of Dr. Sambon's last letter to Dr. Manson, all five had remained in perfect health, while their neighbors were all ill with fever or had suffered malarial attacks. Whereas this experiment had not been completed, those carrying it out were fully convinced that protection from mosquito bites protects from malaria, and that protection from mosquito bites is perfectly compatible with active outdoor occupation during the day.

Manson states that these experiments and those of other investigators indicate that the practical solution of the malaria problem lies (1) in avoiding the neighborhood of native houses; (2) in the destruction, so far as practicable, of the breeding pools of anopheles, and principally (3) in protection from mosquito bites.—*The Lancet*, September 29, 1900, p. 923.

Diphtheritic Paralysis and Antitoxin.—The assertion has occasionally been made that cases of post-diphtheritic paralysis are more frequent now than they were in the days before diphtheria antitoxin. RANSOM, in Behring's laboratory at Marburg, carried out a series of experiments on guinea-pigs with diphtheria toxin and antitoxin with the object of ascertaining whether there was any experimental proof for these statements. It is generally held that the severer forms of diphtheria are most likely to be followed by paralysis, and Ransom states that post-diphtheritic paralysis may be really more common now, owing to the fact that more severe cases of diphtheria now recover as a result of the general adoption of the antitoxin treatment. The technique adopted cannot be here given, but Ransom's conclusions with the use of toxins 79A and 100 are as follows:

1. Paralysis may certainly be expected after intoxication with not less than one-fourth of the minimum dose. With doses between one-fourth and one-eighth paralyses occur, but are not constant, and below one-eighth no paralysis was noticed.

2. The larger the dose of toxin the severer will be the paralysis, if the animal survives long enough.

3. Neutralized mixtures of toxin and antitoxin containing only about one lethal dose or less do not appear to cause paralysis.

4. Antitoxin given fifteen to twenty-two hours after intoxication, with doses of toxin not greater than the lethal dose, exercises in large doses a mollifying influence on the subsequent paralysis. This influence is more evident with smaller doses of toxin than with such as are but little less than the minimum dose. Small doses of antitoxin have no evident effect in diminishing the paralysis.

5. Transferring these results to practice among human beings, we may expect liberal doses of antitoxin, given early in the illness, to influence favorably the subsequent paralysis, and this beneficial influence is likely to manifest itself not so much on the local paralyses (soft palate, etc.) as on such symptoms as failure of the heart. Severe cases are, however, likely to be followed by some paralysis in spite of even large doses of antitoxin.—*The Journal of Pathology and Bacteriology*, July, 1900, p. 397.

SURGERY.

UNDER THE CHARGE OF

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The Causes and Treatment of Movable Kidney.—MOULIN (*British Medical Journal*, March 10, 1900), in considering the causation of movable kidney, speaks as follows: The width of the lumbar recess is one of the penalties which have followed the assumption of the erect attitude. If the history of the development of the renal organs in the race as well as in the individual is worked out it is seen that originally they were distributed over the whole length of the head and body, and that by degrees, as their function became more specialized, they became concentrated together, until in their most perfect form they constitute a pair of organs which in the quadrupedal mammalia are placed in a position of the very greatest possible security.

They lie close to the spine, immediately under the vertebra, almost in the central axis, around which all movements of the trunk and limbs take place. They are buried in two deep and narrow recesses, protected by the lower ribs, and rest upon a horizontal sheet of fascia, which in turn rests on the peritoneal viscera. The slight degree of backward and forward movement which is necessitated by the action of the diaphragm takes place without the least risk; but with the assumption of the erect attitude a great portion of this security is lost. The backward and forward movement is altered to movement upward or downward in the direction of the line of gravity. The peritoneal viscera and the supporting fascia lie in front and not below. The lumbar recesses, instead of being deep and narrow, become broad. The convexity forward of the lumbar spine helps to make them still more shallow. The pelvis, especially in the female sex, becomes wider, so that the lower end of the recess becomes more open, and then the development of right-handedness causes the transverse processes of the lumbar vertebræ, which lie under the floor of the lumbar recess, to rotate. From a position of the greatest safety the kidneys are placed in one from which all security is gone; and it is no wonder that comparatively trivial causes —an increase in the weight of the organ without a corresponding increase in the bulk, a sudden, violent jerk, or a great lowering of the intra-abdominal pressure—can increase their normal range of mobility. The wonder is not that movable kidney occurs, but that it does not occur more often.

This accounts completely for the peculiar clinical distribution of movable kidney. It is certainly hereditary and common. It is present in women much more often than it is in men, and in women who have borne children more often than in those who have not, and it is much more common on the right side than on the left. None of the other reasons which have been assigned for this, such as the weight of the liver or the length of the renal vessels, deserve consideration.

In regard to treatment Moulin says: The choice lies between wearing an abdominal belt and nephrorrhaphy. In the author's experience it is only in the milder cases that the former succeeds, and then it must be combined with massage and exercises calculated to strengthen the abdominal muscles. A belt braces the viscera together and so steadies the displaced organ, but it cannot press the kidney back into its place or retain it there after it has been reduced if the patient stands upright and takes a deep breath. Pads are useless. For all cases in which there is manifest deformity of the lumbar region, associated with movable kidney, or in which there is real distress, even though the mobility attains no higher degree than the antiversion of Potain, he never hesitates to recommend nephrorrhaphy. In the former case it is advisable in order to save the kidney from hydronephrosis and other troubles. It is the most certain method of giving relief. He has never known it to fail, when properly carried out, if the symptoms have not already lasted so long as to produce an indelible impression upon the patient's nervous system.

The Open or Operative Treatment of Fresh Fractures.—SCUDDER (*Boston Medical and Surgical Journal*, March 22, 1900) has made a careful study of the recent and remote results obtained in the treatment of fractures of

the lower extremity. In discussing these results he says: "The treatment of closed fractures has always been thought to be easy and simple. The tables are to-day being reversed; it is the simple fracture which to-day is found to be the most difficult to treat if the problem of treatment is fairly faced. Is there anything more difficult to treat satisfactorily than the simple oblique fracture of both bones of the leg above the ankle; than fracture of the surgical neck of the humerus, with or without displacement of the head of the bone; than fracture of the clavicle, with considerable displacement? Is the separation of the upper epiphysis of the humerus an easy lesion to treat satisfactorily? Instances may be multiplied until it is evident that each bone is frequently fractured in a way most difficult for easy and satisfactory treatment by existing methods.

"The Röntgen ray provides through the developed plate and fluoroscope accurate anatomical knowledge of the relative position of broken bones. The interpretation of the fracture seen through the fluoroscope and upon the plate must be made by one skilled in the art. The casual observer makes many errors in judgment, and misinterprets the conditions present. The exact conditions of exposure and position under which the skiagraph is taken must be known before even the trained interpreter can correctly make the diagnosis. The X-ray provides the means for an accurate diagnosis.

"The open treatment of fractures should be undertaken only after careful consideration of the health of the patient and under most rigid antiseptic and aseptic conditions possible. The open treatment of the fracture should be carried out only by a skilled and competent surgeon. It should not be done in the aged. It is an operation for young adults, particularly for the laboring man. Early massage and passive and active motion after four weeks are desirable in the treatment of the fracture after operation.

"It is impossible to determine which method of those in use to-day will prove effective until the fracture is exposed to view. That method in any given case is the best which preserves the alignment of the shafts of the bones.

"The difficulties of reducing fractures will become more and more apparent as the open treatment is more commonly used. It will be found that no one factor is the cause of the difficulty, but more often it is due to a shortening of all the soft parts of the limb about the fracture, occasioned by the hemorrhage into the tissues, by retraction of tissues which have been normally stretched after the fractures have no resistance offered to their reaction. This difficulty in reduction I have found to be more common than is generally supposed. A recognition of it as a cause will lead undoubtedly to the supplementing of splints by internal fixation in the treatment of many fractures.

"The ideal result to be aimed at after a fracture is union of the fracture without deformity and without impairment of the function of the limb, either immediately or remotely. The generally accepted methods of treating fractures do not give satisfactory results in many cases. There is need for a radical departure in the treatment of closed fractures. Anæsthesia and the X-ray afford the means of accurate diagnosis. Sepsis is practically abolished. From analogy in excisions and osteotomy, closed fractures of bone

may safely be treated by open incision. The cases reported in the literature, expressing the opinions of many surgeons, demonstrate that the open method of treatment is satisfactory and a great advance upon generally accepted methods."

Fracture of the Metatarsal Bones by Indirect Violence.—LOISON (*Revue de Orthopédie*, September 1, 1900), after reviewing the symptomatology, diagnosis, and treatment of thirty-two cases, notes the following conclusions: (1) Complete or incomplete fractures of one or several of the metatarsal bones are much less rare than is generally believed. (2) The X-rays are of the greatest assistance in the diagnosis of incomplete fractures, in establishing the form and exact situation of the fragments, and also the extent of the injury in the complete and comminuted fractures. (3) Fractures of the metatarsal bones as the result of indirect violence are due to either one of two mechanical causes: exaggerated flexion in a vertical direction or in a transverse direction. In some cases these two causes may be combined, the causative traumatic force following an oblique direction.

A Study of the X-ray Plates of One Hundred and Forty Cases of Fracture of the Lower End of the Radius.—CODMAN (*Boston Medical and Surgical Journal*, September 27, 1900) states that Colles' fracture may be separated into ten distinct types, according to the lines of cleavage and the directions of the displacement. Class I. Fracture through the base of the styloid process of the radius. The fragment is displaced but little—generally upward and backward by the supinator longus. Four cases are included in this series. Class II. Fracture of the inner angle of the lower end of the radius. The fragment either remains in place or may be dislocated backward. This type includes three cases. Class III. Transverse fracture at or a little above the epiphyseal line (in adults) without displacement. This class includes sixteen cases. Class IV. The distal fragment is comminuted, either as a simple T-fracture or into several smaller pieces. The fragments are generally displaced posteriorly and radially, but part of them may go anteriorly. In the thirteen reported cases all were complicated by fracture of the styloid process of the ulna. Class V. Separation of the epiphysis of the lower end of the radius. The cases, seventeen in number, presented themselves with the fragments completely reduced, or else they had the typical silver-fork deformity. Class VI. Separation of the epiphysis of the lower end of the radius with a chip off the posterior surface of the diaphysis. This class includes ten cases. Class VII. Impaction of lower fragment into the shaft. Rare; only two of the 140 cases being found in this class. Class VIII. Typical Colles' fracture, which may be divided into two forms: That with marked radial displacement of the fragment and that in which the posterior deformity is more decided. These two deformities are usually combined to a greater or less extent. Of the 140 cases 64 are included in this class. Class IX. Stellate fracture of the lower end of the radius, with longitudinal fissures extending into the shaft. There is but slight deformity. This class includes six cases. Class X. Reversed Colles' fracture—that is,

anterior displacement of the lower fragment. This is almost exactly the reverse of Class VIII., and five cases are included in this series. After describing the diagnosis and treatment of these various types of cases, Codman states in conclusion: (1) That true Colles' fracture formed but 46 per cent. of the 140 cases of fracture of the lower end of the radius. (2) That a knowledge of the position of the fragments is necessary for intelligent treatment. (3) That the application of padding and splints should vary according to the displacement shown in the skiagram. (4) That if the X-ray shows that marked deformity is still present after the splints have been applied, ether should be given and another energetic attempt at reduction made. (5) That concomitant fracture of the ulnar styloid occurred in at least 62 per cent. of the 140 cases, and if 39 cases in which this process was obscure in the skiagram are deducted, fracture occurred in 86 per cent. (6) That in Colles' fracture pressure over the anterior edge of the upper fragment should be avoided and cross traction should be used to correct the radial displacement of the lower fragment. (7) That statistics of the results of Colles' fracture are not of value unless the pathology of each case is determined by a skiagram.

The Treatment of Tuberculous and Purulent Joints with Large Glass-speculum Drainage and Pure Carbolic Acid, with a Report of Seventy Cases.—PHELPS (*New York Medical Journal*, September 15, 1900) states that the belief that interference with purulent or tuberculous foci will result in constitutional infection is not borne out by clinical observation, for he states that he has not only never seen it, but he does not believe that it ever occurs. There should be absolutely no difference between the treatment of abscesses of a joint and that of an abscess occurring in any portion of bone or in soft parts. As pure carbolic acid cannot be absorbed it is of interest to see how it reaches the bacteria that have found their way into the lymphatics remote from the joint. The explanation is founded on the theory that carbolic acid unites with the albumin of the tissues, forming an albuminoid or albuminate which is a powerful antiseptic. This new compound is absorbed into the lymphatics and destroys the bacteria. The method of application consists in exposing the abscess cavity by a free incision, then enlarging the opening into the capsule and explore the joint. If there is extensive bone disease the incision is lengthened and the capsule of the joint freely divided for one-half or two-thirds of its entire circumference, the head of the bone pulled out from the socket, the curette freely used, and the joint thoroughly irrigated with a 1 : 1000 bichloride solution. The joint is now filled with pure carbolic acid. It is allowed to remain one minute by the watch, after which the joint is thoroughly washed out with pure alcohol, and finally the alcohol is washed away with a 2 per cent. solution of carbolic acid. The largest size glass drainage-tube that the cavity will take is then introduced and is retained in place until granulations fill in from the bottom of the wound. The results have been uniformly good. Twenty of the seventy operations were excisions, and these patients were discharged from the hospital in an average of three weeks from the time of operation, wearing hip braces or using crutches.

PEDIATRICS.

UNDER THE CHARGE OF

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The Premonitory Stage of Scarlatinal Nephritis.—In a valuable contribution to the subject of the treatment of scarlatinal nephritis, ROBERT COLEMAN KEMP, of New York (*Pediatrics*, October 1, 1900, p. 241), devotes a section to the urinary indications of the premonitory stage of the renal complication.

1. The first symptom often noted is, as a rule, a fall in specific gravity, without, as yet, the appearance of casts, blood, albumin, or, as a rule, rise of temperature. Anasarca, as a rule, does not appear at this stage of the disease. This symptom, which was first called to the author's attention by Dr. William H. Thomson, of New York, has been observed in many cases without polyuria and without albuminuria, yet in some with commencing anasarca associated with it. Other observers speak only of a fall in specific gravity as a primary symptom and as invariably associated with polyuria, and more or less frequent micturition and irritability of the bladder. The specific gravity should, therefore, be accurately taken at least twice a day from the middle of the second week, and the daily quantity of urine should be measured. Active treatment should be begun the instant a fall in specific gravity is observed.

2. The next symptom following the fall in specific gravity is a sudden rise, becoming abnormally high. The urine is diminished in quantity and is high-colored. Frequently these are the first symptoms noted by the physician, no preliminary fall of specific gravity occurring. As a rule, there is yet no albumin, usually no anasarca, and no exacerbation of temperature, a certain degree of fever generally existing at the time, attributable to other causes.

These signs are especially significant during the period from the second to the fourth week, denoting an engorgement of the renal vessels, and call for active treatment. Occasionally there may be a trace of albumin and a slight rise of temperature, or even a slight anasarca coincident with the above symptoms.

3. The increase of urates, especially with an accompanying slight rise of temperature, especially in the third or fourth weeks of scarlatina, though without albumin, is indicative of a commencing nephritis.

4. As a rule, a period of perfectly normal urine intermits before the development of nephritis. This is the experience of Forchheimer.

[The importance of systematic observation of the specific gravity of the urine during the later weeks of isolation, as pointed out by the author, can-

not be too strongly emphasized. The nurse should, therefore, be instructed in the use of the urinometer in the sick-room, and two daily observations should be recorded with the same regularity as is practised with pulse, temperature, and respiration records.—ED.]

Hysteria in Boys and Youths.—ARTHUR HALL (*The Quarterly Medical Journal*, August, 1900) reports a series of interesting cases illustrating the great danger of mistaking hysteria for organic disease, especially when the patient is a boy.

Case I., a boy, aged nine years, was first afflicted five weeks before observation. He had complained of pain in the legs on returning from school, and two weeks later he began to stagger so that he nearly fell into the fire. Since then he had steadily grown worse, his gait resembling that of cerebellar disease. He lay curled up in bed, objecting to both light and interference. The muscles of the legs and thighs were well developed and firm, with good power, no anæsthesia, complete control of sphincters, and normal reflexes. Perfect cure resulted after an exhibition of the faradic battery and its threatened use.

Case II. was observed in a boy, aged thirteen years, who thirteen days before consultation had sustained a blow upon the frontal region of the head from a falling box. No external lesion had been produced. The same day he had a fit, and continued to have two or three a day since. There was no vomiting; photophobia was complained of; but there was no paralysis, no anæsthesia, and knee-jerks were increased. The ocular fundi were normal. The "boiling-water" treatment during a very characteristic hysterical attack was very successful. In this expedient boiling water is loudly called for; cold is supplied, and, if necessary, is sprinkled on the brow.

Several other cases are recorded, two simulating cerebral tumor, in a boy of nine years and a youth of eighteen years respectively; one simulating meningitis (fifteen years), one simulating Jacksonian epilepsy (twenty-one years), and one simulating a lightning stroke (a youth).

In conclusion, the author remarks that the most striking difference between the hysterical boy and the hysterical girl is that the former apparently does it much more purposely, the difference being partly due to the difference in surroundings of the two. The boy has to live among boys, and knows how his fellows would tease him for any "girlish" tricks, hence he dreads discovery, and "plays the game" with greater thoroughness.

Abdominal Symptoms in the Pneumonia of Children.—J. L. MORSE, of Boston (*Annals of Gynecology and Pediatry*, November, 1899), calls attention to the danger of mistaking a case of so-called abdominal pneumonia in children for one involving the abdominal organs and demanding operative relief. He reports that he has seen two cases in which the abdomen was opened by experienced surgeons, because appendicitis and not pneumonia was supposed to be the cause of the trouble. In such cases physical signs in the lungs may not be recognizable for several days. In the absence of physical signs the combination of symptoms should arouse suspicion of the real seat of the disease. With an acute onset, with high temperature and the rate of respiration increased out of proportion to that of the pulse, even

in absence of a cough, the probability of pneumonia is decided. This disturbance of the ratio of pulse, temperature, and respiration should enable one to avoid error, even when abdominal pain, constipation, and at times also tenderness and distention, probably of reflex origin, are present. Of the illustrative cases one is quite noteworthy, since the history showed previous constipation and a blow upon the abdomen shortly before the onset of the acute symptoms.

Acute Dilatation of the Heart in Influenza of Children.—F. FORCHHEIMER, of Cincinnati (*Jacobi's Festschrift*, May 6, 1900, p. 127), calls attention to the frequency of acute dilatation of the heart in this disease as especially observed in childhood. He describes two classes of cases, differing essentially in the gravity of their symptoms. In the milder cases within twelve to twenty-four hours of the onset, with high fever accompanied by respiratory or gastro-intestinal symptoms, there develops dyspnœa, or, better, tachypnœa, since all other evidences of dyspnœa are wanting, such as cyanosis and movements of the accessory respiratory muscles. The child lies in bed, with congested skin, sleeps a great deal, yet is easily aroused; the tachypnœa is represented by rapid breathing (60 or 70 or more respirations per minute), without disturbance of the normal relation between inspiration and expiration. In some cases the breathing is not very much more rapid than normal. The pulse is very rapid, depending upon the age of the patient, frequently intermittent, but otherwise normal. Physical examination of the chest reveals nothing, or, possibly, only evidences of a bronchitis in the large tubes. The heart dulness is always found broadened, with no bruit as a symptom of this affection. The urine contains no albumin. In the course of from twenty-four to seventy-two hours all these symptoms disappear—the temperature, pulse, heart, and respiration become normal. After discussing the various theories as to the etiology of this condition the author inclines to agree with Sansom, that there is some affection of the bulb involving both the respiratory and the cardiac accelerating centres. The source of this irritation would be a toxin, hypothetical as far as the influenza bacillus is concerned, but positive as to some of the pus producers which habitually accompany influenza. It would be fully in accord with the nervous origin of these symptoms that broadening of the heart's dulness is found. The production of acute dilatation of the heart as the result of disturbance in innervation has been conclusively proven in many diseases.

The grave form of the affection presents an entirely different clinical picture. Of this Huchard gives the following concise description: "The attack manifests itself by syncope and faintness, which may become fatal, by slow pulse, by arrhythmia, or intermittence, by grave symptoms of cardiac collapse, and sometimes by pains resembling angina pectoris."

The dominant symptoms in all the cases among children seen by the author were the acute dilatation and the peculiar respiration which he has seen only in two other conditions—in pericardial effusion and the fugitive œdematous condition of lung that frequently precedes pneumonic consolidation in influenza without cardiac changes. According to his observation, one is justified in assuming two forms of dilatation of the heart in influenza: one presumably produced by the action of the toxin upon the nervous sys-

tem of the heart, and possibly upon the myocardium; the second form occurring in such conditions in which outflow of blood is materially interfered with on account of mechanical conditions. The first form, according to the observations of West, undoubtedly may end fatally, although this has not been the case in the author's own experience. The second form is one that lasts much longer than the first, but in children has a tendency to recovery.

THERAPEUTICS.

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The Treatment of Furunculosis.—DR. GUSTAV LONGMANN applies the cathode, which exhibits more intense reaction upon the tissue. It is placed upon the furuncle, the anode somewhere in the neighborhood, and a current of about 2 to 5 milliampères is applied for from five to ten minutes. As a rule, one sitting daily is sufficient. The intensity of the current ought to vary both with the stage of development of the boil (the earlier stages being more sensitive) and with individual sensitiveness. Whenever there is extensive suppuration a flat pledget of cotton soaked in a 1: 1000 solution of corrosive sublimate is placed between the sponge and the skin. Whether this powerful antiseptic has a beneficial cataphoric action or not is not certain, but those who have great confidence in cataphoresis may use it on all boils. Under this treatment some boils disappear after one or two applications; others may require as many as eight; on the average four or five sittings terminate their existence.—*New York Medical Journal*, 1900, No. 1122, p. 854.

Chronic Brass Poisoning.—DR. WILLIAM MURRAY remarks that while the condition is far from infrequent, the literature is surprisingly scanty and unsatisfying. Potassium iodide did not give satisfactory results in spite of Simons' enthusiastic indorsement. Remembering that copper sulphate is recognized as an antidote for phosphorus poisoning, pills containing the latter in doses as large as one-tenth of a grain daily were administered, with excellent results. The patients became rapidly well, even if they did not desist from their work. Diluted phosphoric acid, fifteen minims three times daily, gave even better results; the explanation being, apparently, that phosphorus when injected becomes converted partly into phosphoric acid, and it may be that only this part is effectual, so that a certain amount of economy is exercised in giving the acid direct. Sometimes, however, better results were obtained with the phosphorus. The only matter of diet which

need be considered is the importance of milk as a beverage.—*British Medical Journal*, 1900, No. 2057, p. 1334.

The Untoward Action of Citrophen.—Dr. Ernst Schotten was treating a young woman for acute rheumatic polyarthritis with sodium salicylate, which produced ear symptoms and vomiting. Immediately on commencing with citrophen in fifteen-grain doses thrice daily there supervened headache, hot flashes, singing in the ears, and profuse perspiration. On the following day cyanosis appeared. Neither at this nor other times was the pain markedly diminished. On omitting the drug these symptoms disappeared.—*Therapeutische Monatshefte*, 1900, Heft v., S. 278.

The Action of Chloroform upon the Blood.—Dott. V. Baccarani presents the following interesting results: (1) The specific gravity of the blood is not altered in mild narcosis; in some species of animals it is increased, in others diminished by protracted and profound narcosis. (2) Its alkalinity is slightly diminished. (3) The number of red corpuscles is diminished, but not always in a given proportion. (4) The number of white corpuscles is increased, particularly in the peripheral bloodvessels. (5) There is no appreciable change in the percentage of hæmoglobin when measured by the Fleischl apparatus. (6) Arterial blood shows the absorption-band of methæmoglobin. (7) The bactericidal properties are increased (8) It furnishes the respiratory power of the red blood-cells (9) Venous blood reacts more readily and more markedly to chloroform. (10) The red blood-cells of youth are more profoundly changed than those of adults. (11) Death may occur in animals without notable change in the blood. (12) The action of chloroform may persist for several hours, or even days.—*Gazetta degli Ospedali e delle Cliniche*, 1900, No. 42, S. 445.

The Treatment of Articular Rheumatism.—Dott. F. Battistini, while regarding the salicylates as the most trustworthy remedies, is not insensible to their disadvantages. Better known, and at the same time inconveniences of minor importance, are digestive disturbances, varying from heaviness in the stomach to nausea and vomiting. Various exanthemata, morbilliform and scarlatiniform erythema, erythema nodosum, urticaria, vesicular pemphigoid eruptions, and, rarely, in marasmic individuals, purpura may appear. Hyperidrosis is noted in about 68 per cent. of patients. Hemorrhage is of comparatively recent occurrence with sodium salicylate—epistaxis, hæmaturia, entorrhagia, metrorrhagia, and menorrhagia. Various nervous symptoms have been observed, notably delirium. Dyspnœa merits especial attention, and is produced by direct action on the respiratory centres. Symptoms referable to audition occur in about 60 per cent. of urticarias. For diminishing the last ergot or ergotin have been proposed, but do not give satisfactory results. For preventing gastric disturbance weak solutions of the remedy should be employed, or alcohol added in small quantity; or the remedy may be associated with sodium bicarbonate or Vichy and given apart from the meals. Many instances of intolerance can be circumvented by rectal administration, high enema, well diluted, and with a small amount of tincture of opium.—*Rivista Critica di Clinica Medica*, 1900, No. 17, p. 331.

Acetanilid Poisoning.—Dr. O. R. Summers reports a patient who was given four grains and the dose repeated in thirty minutes. There was no cardiac lesion; the patient suffered from temporary frontal headache. Twenty minutes after the second powder was taken there developed nausea and giddiness. Headache suddenly ceased; respiration became very difficult, 35 to the minute; pulse, 140; temperature, 95° F.; cyanosis of lips and fingers; cold extremities; dilated pupils; cold perspiration; convulsive movements; partial loss of consciousness; retching. Whiskey and strychnine nitrate were given; artificial respiration was practised for nearly two hours. The patient rallied, but relapsed and nearly died. Renewal of the above treatment again caused improvement in her condition and complete recovery ensued. For four days she was confined to bed, with slight fever and considerable vertigo. It is of importance to note that the patient had taken much larger doses of acetanilid many times with no ill effects, showing that there was no idiosyncrasy for the drug.—*New York Medical Journal*, 1900, vol. lxxi., p. 426.

Treatment of Scarlatinal Nephritis.—Dr. Robert Coleman Kemp recommends amyl nitrite by inhalation for uræmic convulsions. As regards the use of oxygen in pulmonary conditions due to nephritis may be cited pulmonary œdema, pulmonary congestion, or pleurisy with effusion due to oliguria, as sufficient examples The early use of this agent is the keynote of success, for it will dilate the bronchioles, improve the respiratory functions, and also the pulmonary and general circulation. Such results must diminish the chances of further extension of the morbid processes, and aid the absorption of those products if already present. As an aid to the elimination of toxins, it improves the condition of the red blood-cells and the secretory functions. One of the most valuable agents is normal saline solution. Enteroclysis at 110° to 120° F. with this solution for fifteen minutes to an hour continuously, depending upon indications, and as frequently as three or four times daily in oliguria, uræmic convulsions, uræmic coma, dropsy, suppression of urine, pulmonary œdema, pleurisy with effusion, gives satisfactory results.—*Pediatrics*, 1900, vol. x., pp. 241, 281.

Anthrax Successfully Treated by Local Injections of Pure Carbolic Acid.—Dr. W. E. Fisher reports a single instance in which one drachm of a 10 per cent. solution of carbolic acid was injected into and around the eschar of a punctured wound on the forearm, received eight days before the patient came under observation. Injections of one drachm of pure carbolic acid were repeated on the following days until six, in all, were given. After three injections the temperature fell to normal, and after five the eschar ceased to spread. At no time was carbolic acid detected in the urine. —*Therapeutic Gazette*, 1900, No. 8, p. 508.

Tansy.—Dott. Nicola Lanza has made a laboratory study of this plant. The following conclusions are presented: 1. In frogs after injection of small or moderate doses of the essence of tansy there is, first, a temporary period of excitement, due in part to the local irritant action of the drug,

then follows a progressive weakening of motor power, and finally a complete loss of voluntary movement and a diminution of the reflexes. With large doses there comes a period of excitement, followed by complete loss of motion and sensation, paralysis, muscular resolution, and death. 2. In guinea-pigs and rabbits, with small and medium doses, tremors, hyperæsthesia and diminution of voluntary movements follow. With large doses we have first isolated spasms of the muscles of the face, thorax, and anterior extremities, then general tonic-clonic convulsions, loss of voluntary movement, and death. 3. In rabbits are observed the classical *rabies tanacetica* of Peyraud—grinding the teeth, attempts to bite the foot, and chewing their paws. 4. In dogs after subcutaneous injection of small or moderate doses there follow local irritation, renal irritation, with bloody urine, and tremors. With large doses these symptoms are accentuated with consecutive paralysis. 5. After intravenous injection follow convulsive attacks, at first tonic, but later tonic-clonic, paralysis, coma, and death. 6. In dogs the paralytic form of tansy-rabies prevails. 7. In pigeons small doses produce a temporary excitement; large ones, general tonic-clonic convulsions, and death by asphyxia.—*Gazetta degli Ospedali e delle Cliniche*, 1900, No. 9, p 81.

The Electrostatic Treatment of Paralysis.—Dr. William Benham Snow summarizes as follows: 1. In an instance of paralysis the nutrition of the parts should be maintained by an agency which will preserve the balance of the nutrition of exercise and the nutrition of restoration, that degeneration may be retarded. 2. Such agents must be searching, potent, and of a character which will induce no chemical or unfavorable alterative action of the tissues. 3. Use electrostatic sparks and currents, having high voltage and infinitesimal quantity or ampèrage, exercise the parts, promote metabolism, and, when systematically applied, thereby one maintains the parts in the best condition. 4. When the character of the lesion does not preclude the possibility of recovery, early treatment will prevent degeneration, hasten recovery, and give excellent final results. 5. There is not any contraindication, at any state of a paralysis, to the intelligent administration of electrostatic treatment, because it lessens hyperæmia and congestion, relieves nervous irritation, and tends to restore the normal equilibrium of the system in general and of the parts affected.—*The Post-Graduate*, 1900, No. 3, p. 358.

Night-terrors.—Dr. Francis Huber would proscribe threats, ghost stories, goblins, giants, the usual thrilling nursery literature, exciting games, and dark or poorly-ventilated rooms. Heavy, indigestible meals at night and the use of tea, coffee, or alcohol in any form favor the occurrence of this symptom in those predisposed. The causes being varied, each patient must be studied. The mode of life must be investigated, the habits inquired into, nervous or mental strain avoided, and the influence of parents, companions, and attendants subjected to close inquiry. The literature, games, and diet are worthy of attention. Pale, anæmic, and weakly children must be strengthened by tonic treatment. The presence of some person in an adjoining room and a dim light may increase the confidence of the child. In neuropathic children the bromides or chloral may render good service.—*Pediatrics*, 1900, No. 7, p. 279.

GYNECOLOGY.

UNDER THE CHARGE OF

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Separation of the Corpus Uteri.—LUDWIG (*Centralblatt für Gynäkologie*, 1900, No. 25) reports a case of ovariotomy in a child, twelve years of age, in which the uterus was found to be absent from its usual site. After a search it was found to be drawn upward and attached to a dermoid cyst, a thin band of tissue being traced downward to the vaginal vault. The organ was well developed, the tubes occupying their normal relations to the cornua. The cervical canal was obliterated, though the corporeal cavity was of the usual size. On vaginal examination a small portion was found with a canal 3 mm. in length. It was evident that the body of the uterus had been gradually separated from the cervix by upward traction made by the growing cyst.

Implantation of the Fallopian Tube in the Uterus.—SCHMIT (*Centralblatt für Gynäkologie*, 1900, No. 25) in performing a cœliotomy for tubal pregnancy, found the ovary normal, while at the proximal end of the tube was a small fibroma which apparently closed its lumen. In order to prevent subsequent sterility or a possible second tubal gestation, the operator excised the affected portion of the tube and implanted the end in the uterine cavity. The patient made an uninterrupted recovery.

Etiology of Uterine Fibromyomata.—MÖLLER (*Centralblatt für Gynäkologie*, 1900, No. 25) discusses at length the two theories with regard to the etiology of these growths, viz., that they are due to local irritation, or that they are due to prenatal causes. From an analysis of a large number of clinical cases he finds that 32.8 per cent. of the patients were virgins, and 67.2 per cent. were not. About one-half of the latter were sterile. Heredity seemed to play quite an important part. In his microscopical studies the writer often found myomata the size of a pin's head, which were separated from the uterine muscle by a distinct layer of connective tissue. Small arteries could be traced into the periphery of the interstitial growths from the surrounding tissues. The vessels possessed the usual three coats, though the intima and media eventually disappeared. The muscle-cells in the vessel walls remained distinct from those of the myoma, which the writer believes is an argument against the theory that these growths may originate from the muscular elements of the bloodvessels. Personally, he believes that they may be produced by local irritation in a subject with a predisposition to these tumors.

Restitution of the Pelvic Floor.—OTT (*Centralblatt für Gynäkologie*, 1900, No. 28) describes the following method of operation in a case of prolapse of the rectum due to complete atrophy of the muscles of the pelvic floor, in-

cluding the sphincter ani. The skin carrying the perineum was dissected off, after which a denudation was made similar to that in cases of complete laceration. The sutures were passed in such a way that the lower portion of the rectum was narrowed and somewhat lengthened. The operation was entirely successful, and an examination at the end of three weeks showed that the normal tone of the pelvic floor had been restored, and that a firm, elastic sphincter muscle was present.

Results of Vagino-fixation.—KAUFFMANN (*Zeitschrift für Geb. u. Gynäkologie*, Band xlii., Heft 1) reports the subsequent condition of 103 patients upon whom this operation had been performed at the Berlin Clinic. In all but three, plastic operations upon the vagina and pelvic floor were done at the same time. Only such cases were treated surgically as could not be relieved by pessaries. The periods after operation varied from eight months to four years.

In eleven cases the retroversion recurred, but in only one of these had the peritoneal cavity been opened. The fact that prolapse of the vagina was almost invariably present led to the inference that the uterine displacement followed the sagging of the vaginal walls. Five patients became pregnant after the operation, four being delivered at full term. All but seventeen of the patients had passed the age of child-bearing. The writer emphasizes the importance of limiting vagino-fixation as far as possible to such cases, and believes that shortening of the round ligaments or ventro-suspension should be preferred in women who are not near the climacteric. Plastic operations should always be performed at the same time if the vagina is relaxed.

Displacements and Chronic Disease of the Adnexa due to Physical Strain.—FÜRST (*Sammlung klin. Vorträge*, No. 253) states that retroversion is favored by a moderate increase in the intra-abdominal pressure, if the latter is more or less constant. Seamstresses are peculiarly liable to this condition, as well as young girls who are obliged to sit for long periods in an unnatural position while studying. Excessive cycling is also a frequent cause. Women whose occupations favor a continued diminution of the intra-abdominal pressure, such as scrub- and washerwomen, seem to possess a certain immunity. The writer advises patients with a tendency to retrodisplacement to assume the knee-chest position for at least ten minutes daily. Disease of the adnexa is favored by constant severe muscular efforts, such as carrying burdens or running heavy sewing machines. The greater frequency of parametritis in the left side he attributes to the more frequent use of the right arm, which causes the intra-abdominal pressure to be exerted toward the left side of the pelvis.

The Clinical Significance of Movable Retroversion.—KRÖNIG and FENCHTWANGER (*Monatschrift für Geb. u. Gyn.; Centralblatt für Gynäkologie*, 1900, No. 28) analyzed a series of cases of anteflexion and retroflexion, with the view of determining which displacement occasioned the more severe symptoms. 103 cases of the former and 33 of the latter were kept under observation, 44 per cent. of the women with anteflexion and 40 per cent., or

those with retroflexion, complained of practically the same troubles, which seemed to show that the position of the uterus has little to do with the symptoms. Hysteria seemed to be the most frequent one in retroflexion, but the relation did not appear to be close. A further study of cases in which the displacement had been curetted by pessaries or operations also seemed to bear out the conclusion that the persistence of the symptoms was not due directly to the better position of the uterus. The authors infer that cases of movable retroversion with attendant nervous symptoms are really subjects for medical treatment.

Precautions after Narcosis.—BLUMFELD (*Lancet; Centralblatt für Gynäkologie*, 1900, No. 28) makes the following valuable suggestions, which are especially applicable to cases of abdominal section: 1. At the conclusion of the operation the stomach should be washed out if the patient has swallowed considerable mucus. 2. If an operation has continued beyond three-quarters of an hour ether should be substituted for chloroform. 3. The patient should be moved as little as possible during and after operation. 4. She should be placed upon the right side. 5. Nothing but small quantities of hot water should be given for at least eight hours. 6. The temperature of the room should remain the same for several hours. By observing these precautions the writer found that in 572 narcoses one-third of the patients remained entirely free from unpleasant after-effects.

Tympanites as a Diagnostic Sign.—BAYER (*Prager med. Wochenschrift*, 1899, Nos. 25, 26, and 28) believes that the character of the tympanites in a given case gives a clew to the location of the obstruction in the intestinal tract. He distinguishes the following types: 1. Dilatation of the stomach due to pyloric stenosis, which in obscure cases may be accentuated by artificial distention of the organ. 2. Stenosis of the duodenum, in which there is constant vomiting of bile without symptoms of ileus. 3. Distention of the whole epigastrium, with flattening of the lower abdomen and ileus, points to obstruction of the upper portion of the small intestine; in some cases the tension is suddenly relaxed after severe retching. 4. Prominence of the umbilical region, with depressed flanks, is indicative of stenosis at Bauhin's valve. 5. Obstruction low down in the large intestine is attended with distention in the flanks and broadening of the lower abdomen; if higher up the distention of the colon is partial or asymmetrical. The greatest tension in the cæcal region occurs in cases in which the obstruction is below this point. The writer adds that these typical forms of meteorismus are only valuable for diagnosis so long as the peristaltic movements still persist. The diagnosis becomes more difficult in general paralysis of the gut due to peritonitis. Errors may easily arise when coils of adherent intestine are so situated as to resemble neoplasms; also in fecal impaction.

Superficial adhesions are attended with the frequent development and rapid disappearance of painful sausage-shaped swellings accompanied by cramp-like pains.

Multiple stenoses offer the greatest difficulties.

OBSTETRICS.

UNDER THE CHARGE OF

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The Influence of Narcosis by Chloroform upon the Pains of Labor.—WESTERMARK publishes in the *Archiv für Gynäkologie*, 1900, Band lxi. Heft 1, the reports of a series of elaborate experiments to determine the influence of chloroform upon the action of the uterus during labor. These experiments were conducted in Stockholm, and are of great interest. He concludes that complete narcosis by chloroform diminishes the frequency of uterine contractions and lengthens the interval between the pains. It exercises no influence upon the intra-uterine pressure between the pains, but renders uterine contractions shorter in duration. The intra-uterine pressure is lengthened during the pain under the full influence of chloroform. The pain reaches its highest point in practically the same time as in cases where chloroform is not given. Obstetric anæsthesia with chloroform lessens the frequency of the pains. It lengthens the interval between them, exercises no influence upon the intra-uterine pressure during the pains, but greatly diminishes the suffering which the pains occasion. In a small degree obstetric anæsthesia with chloroform lessens the duration of the pains. It does not, however, diminish the intra-uterine pressure. The highest point of the pain is reached in about the same time with or without obstetric anæsthesia. The practical conclusion of his experiments is that the use of chloroform in obstetrics should be limited to those cases where the suffering is intense and the control of the patient becomes so necessary that it seems best to risk some prolongation of the labor in order to control them.

The Notification of Cases of Puerperal Fever.—At the recent meeting of the British Medical Association, BERRY HART (*British Medical Journal*, 1900, No. 2072) read a paper upon the subject of puerperal fever, of which he distinguished three forms. The first is the acute and rapid form, where a large amount of poison is directly absorbed, either through extensive lacerations or through the retention of placental or membranous tissue. In these cases the pulse may be high while the temperature is low, and the patient has the intoxicated appearance which indicates the gravity of the condition.

The second form is the ordinary one, where the pulse and temperature rise within the first five days, with rigors and invasion of the peritoneum, pericardium, or endocardium. A large number of these cases recover with appropriate antiseptic treatment.

The third variety, sometimes called the venous form, is that in which thrombi become infected and infected material is carried extensively through

the circulation. Pyæmia subsequently develops. In addition to these we see gonorrhœal cases and puerperal tetanus, caused by the tetanus bacillus.

It is urged that these cases be reported to the authorities, just as scarlatina, diphtheria, and other contagious and infectious maladies are reported. It is hoped by this means that puerperal fever may be reduced in frequency, and that important statistics may be gathered which will throw new light upon this disorder.

Cœliohysterectomy for Contracted Pelvis.—In the *Scottish Medical and Surgical Journal*, September, 1900, BREWIS reports the case of a patient whom he saw in labor and in whose case craniotomy was performed. The patient recovered and subsequently became pregnant again. She had a symmetrically contracted pelvis of a high degree of contraction. She assented to any operation which would save her child and would spare her the possibility of further pregnancy. Accordingly, the child was extracted by Cæsarean operation. The ovarian arteries and vessels accompanying the round ligaments were tied and the broad ligaments divided. A peritoneal flap in the lower part of the uterus was pushed down along with the bladder. The uterine arteries were then secured and the body of the uterus was removed. The stump was sutured and covered by peritoneal flaps. The patient made an excellent recovery.

Cephalotripsy for the Aftercoming Head.—At the last meeting of the British Medical Association, TARGETT described the case of a patient, aged twenty-nine years, a rhachitic dwarf. The pelvis was highly contracted, the true conjugate not exceeding two and three-quarter inches. The patient declined to submit to Cæsarean section, and labor was induced. There was entire lack of action on the part of the uterus. Dilatation was performed by elastic bags, version was done, and the breech brought down into the cervix, but without exciting uterine contractions. It became necessary to deliver, when it was found that the head could not pass through the brim. The occiput pointed to the right, and the face was to the left, and flexion was well performed by traction upon the jaw, yet delivery could not be accomplished. Accordingly, the head was perforated in the suboccipital region. The lower blade of the cephalotribe was then applied over the face, but it was impossible to pass the upper blade over the occiput. Accordingly, the upper blade was passed through the suboccipital perforation and the head crushed. The occiput was rotated in front and the head delivered without further difficulty. The patient made a good recovery.

He also described a case in which perforation and cephalotripsy had been performed, but the head was high in the pelvic cavity and the trunk would not enter the brim. Internal version was performed and the child extracted with difficulty.

In the discussion others related cases in which version after craniotomy was most successful.—*British Medical Journal*, 1900, No 2072.

The Second Stage in the Formation of the Human Placenta.—In the *British Medical Journal*, 1900, No. 2072, TUSSENBROEK gives the following results from her investigations in this subject. The macroscopical form of

the placenta is completed about the sixth month of pregnancy. At that period the decidua reflexa has almost totally disappeared. The reduction of the decidua reflexa is the effect of mechanical pressure. The villi of the chorion, which disappear, are removed chiefly by the obliteration of the intervillous spaces between the chorion and reflexa.

The Prevention and Treatment of Post-partum Hemorrhage.—This subject was discussed at the recent meeting of the British Medical Association, the basis for the discussion being a paper contributed by BYERS, of Belfast (*British Medical Journal*, 1900, No. 2072). It is stated that the average amount of blood lost in childbirth is about one pound. Clinically, however, no fixed amount can be taken as constituting hemorrhage, as each patient differs in this respect from others. The obstetrician must expect that post-partum hemorrhage may occur where there is a history of hemorrhage at previous confinements, where pregnancies follow each other rapidly, where patients take little exercise, eat freely and use stimulants, in elderly primiparæ, and where metritis exists during pregnancy. Overdistention of the uterus or the presence of a tumor also favors hemorrhage. Albuminuria, extreme mental depression, and disturbances of the vascular system predispose to it. During labor, pains which are strong and quick, but cease suddenly, with long intervals between the pains, should arouse suspicion of uterine exhaustion; rapid, jerking pulse, with low tension, is also present in many of these cases.

In prophylaxis, attention is called to the importance of managing properly the third stage of labor. The left hand of the obstetrician must follow down the uterus, not using massage or stimulating it in normal cases, but controlling it. The cord is tied by two ligatures—one near the child and the other close to the vulva of the mother. When the placenta has been separated several inches of the cord are expelled; this can be determined by the altered position of the ligature upon the cord. The separation of the placenta is also recognized by a swelling detected above the pubes, due to the bulging of the lower uterine segment, and sometimes taken for a distended bladder. The uterus rises up suddenly and becomes more movable than formerly.

In addition to the proper delivery of the placenta and the removal of all portions, it is most important in avoiding post-partum hemorrhage not to deliver in the absence of pains. This is almost sure to be followed by severe hemorrhage. Byers counsels the use of strychnine and ergot before and during labor in suspected cases, with hydrochloric acid in plethoric patients and iron in those who are anæmic.

In the actual treatment of hemorrhage he would use uterine massage, hot intra-uterine douching with normal salt solution, the introduction of the hand and the removal of adherent portions of placenta or membrane, packing the uterus with antiseptic gauze, and dragging down the uterus, after gauze packing, by stout tenaculum forceps passed through the lips of the cervix. Should hemorrhage occur from lacerations they must be closed by suture. In after-treatment the injection of normal salt solution and stimuli, with abundant nourishment, is indicated.

In discussion, BOXALL thought the loss of blood in labor to be not less

than twenty ounces in the average case. In a considerable number there was no assignable cause for hemorrhage. He had observed that alcohol favored hemorrhage, while rupture of the membranes and prompt delivery prevented it. Quinine and nux vomica given before labor were useful, while ergotole has been of service in his cases.

DERMATOLOGY.

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Tinea Versicolor of the Face.—POWELL (*British Journal of Dermatology*, April, 1900), in a note read before the Dermatological Society of Great Britain and Ireland, calls attention to the frequency of tinea versicolor of the face in Assam, India. In less than three months he saw fifty-six cases, nine of which were examined microscopically, and in all of these the microsporon furfur was found. On black skins the patches are of a chamois-skin color. The fungus seems to be identical with the European variety. The author attributes the frequency of the affection to the fact that the natives anoint the face and body with oil, and seldom use soap.

Lupus Erythematosus and Multiple Carcinoma.—KREIBICH (*Archiv für Dermatologie und Syphilis*, Band li., Heft 3) reports a case of erythematous lupus accompanied by multiple carcinoma. The lupus began upon the lower lip some two years and a half before the patient came under observation; later the upper lip and the nose were affected. About two years later the cancerous affection began. The author regards the case as one of cancer developing upon an inflammatory basis, the inflammation, through œdema and loosening of the connective tissue, making possible the atypical proliferation of the epithelium.

The Histopathology of Pityriasis Rosea.—HOLLMANN (*Archiv für Dermatologie und Syphilis*, Band li., Heft 2) finds that in the macular stage of pityriasis rosea the changes are chiefly in the cutis, and consist of a marked widening of the superficial vascular network, a more or less noticeable perivascular cell-infiltrate in the papillary body and the subpapillary layer of the cutis, and œdema of the upper part of the corium. In a later stage of the disease all these changes are still more marked, especially the perivascular infiltrate. The rete also shows considerable changes: interepithelial and intraepithelial œdema is marked, and there is a proliferation of the prickle-

cell layer, especially in the interpapillary portions. Further on in the course of the affection small vesicles are formed beneath the corneous layer of the epidermis. After a time the horny layer is cast off; with this the disease reaches its acme. The author is of the opinion that the disease process begins in the cutis, and later involves the different layers of the epithelium.

Scleroderma Following Injury.—LESLIE ROBERTS (*British Journal of Dermatology*, April, 1900) reports very briefly the case of a child who, when two years of age, fell against the edge of a fender, wounding the surface of the abdomen. Shortly afterward the bruised surface became indurated, and during the next two years the induration advanced toward the right shoulder in isolated patches separated by healthy skin. Reaching the shoulder it descended the arm, following the cutaneous branches of the median and radial nerves. The index finger became contracted, the thumb stiff, and the skin between the finger and thumb rigid.

The Experimental and Clinical Properties of the Achorion Schonleinii.—BUKOVSKY (*Archiv für Dermatologie und Syphilis*, Band li., Heft 3) concludes from experimental inoculations upon the skin of man and into the animal organism that the quality of the soil plays a great part in the fate of the fungus. The more indifferent the skin shows itself against the invasion of the fungus the more the danger of the occurrence of favus; the stronger the inflammatory reaction the less tendency to the disease, and the occurrence of vesicular inflammation makes the formation of a scutulum impossible. The achorion is not toxic for the animal organism. After its intravenous injection there arises in the lungs the macroscopic picture of a mycotic pseudotuberculosis. Microscopically leucocytic nodules are seen, with giant-cells of epithelial origin, around the threads. The fungus grows only in a rudimentary manner owing to the cell infiltrate hindering its vegetation.

A Clinical and Bacteriological Study of Impetigo.—SABOURAUD (*Annales de Dermatologie et de Syphiligraphie*, 1900, Nos. 1 and 2) presents in two elaborate memoirs some of the results of his study of the several forms of impetigo. He distinguishes two clinical varieties. The first of these is the impetigo contagiosa of Tilbury Fox; the second, the impetigo of Bockhart, characterized by rounded pustules, each containing a hair in the centre and having pustular contents from the beginning. Impetigo contagiosa is a contagious, auto-inoculable malady in which the streptococcus is constantly present. It may assume an ulcerative form, and thus become ecthyma, an affection which has incorrectly been made a morbid entity. Secondary infection by the staphylococcus almost always occurs and causes suppuration. By the pullulation of the staphylococci pustules of purely staphylococcic origin may be produced between the lesions of impetigo contagiosa. In the author's opinion the streptococcus of impetigo is the streptococcus of Fehleisen.

The Histopathology of Epidermolysis Bullosa.—ELLIOT (*New York Medical Journal*, April 28, 1900), who examined sections of skin objectively normal and unirritated, taken from a case of epidermolysis bullosa under his

observation, found the stratum corneum and stratum granulosum normal. In the deeper portions of the stratum spinosum, especially in the interpapillary prolongations, degenerative cellular changes were noticed. The basal layer was the particular seat of the alterations, and was attacked in its entirety. The changes involved the entire cell, altering its staining properties in varying degree. These cellular changes are attributed by Elliot to coagulation necrosis. He believes the chief feature in the morbid process to be an acquired or inherited abnormal irritability of the vascular apparatus of the skin, and, as the consequence of constant response to slight irritation, the basal portion of the rete is continually bathed in serous transudation, bringing about degenerative changes.

Rontgen Rays in the Treatment of Skin Diseases, and for the Removal of Hair.—WILLIAM ALLEN PUSEY (*Journ. of Cut. and Gen. Urin. Dis.*, July, 1900) states that the effect of the rays upon the skin and subcutaneous tissues are all inflammatory in character, ranging from slight erythema to violent inflammation, ending in necrosis. The two actions of the Röntgen rays on tissues which offer the most promising prospects of utilization for therapeutic purposes are: (1) Its power of causing the falling out of the hair, and (2) its power of causing inflammatory reaction and influencing the nutrition of connective tissue. The practical problem is the application of the rays in such a manner that undesirable results be avoided. One should not use a current of more than one and a half ampères and twelve volts' strength, this current, of course, being much weaker than that employed for skiagraphy. An inductor of more than 30 cm. spark-length should not be used, nor should exposures be too long or too frequent. At the beginning the sittings should not be longer than five minutes, and the distance of the tube not more than 15 cm. Freund (who has done considerable experimenting upon the skin) recommends for the attainment of the best results the use of a mechanical interrupter run at the rate of 800 to 1000 interruptions per minute. Suitable lead masks, for protecting surfaces which are contiguous to the areas to be treated, should be used. The evidences that the exposures have been carried far enough are: The appearance of erythema or pigmentation; the blanching of the hair, and the loosening of the hair. The application of the Röntgen rays has been chiefly in four classes of affections: In hypertrichosis, for the removal of hair; in diseases of the hair and of the hair follicles; in inflammatory diseases, like chronic eczema, and in certain specific diseases, like lupus. The author believes that the agent is a valuable one for the removal of hair, especially in cases where the growth is diffuse and profuse. In lupus the rays seem to have a selective action, and, employed properly, constitute a remedy of value.

An Antiseptic Soap.—HERBERT SKINNER (*Brit. Journ. of Derm.*, May, 1900) gives the following formula for an ethereal solution of soap which can be made extempore. It can be rendered antiseptic, not with mercuric chloride, but with the biniodide freshly prepared—that is, mercuric chloride dissolved in a strong solution of potassium iodide. ℞. Acidi oleici, alcohol, of each, ℥ss; liquor ammonii fort., q. s.; ether methylat., ad. f℥ij. The first two are mixed and the ammonia added in order to neutralize the acid, then add the ether. It is far superior to any ordinary soap.

Herpes Zoster of the Finger.—SUERMONPREZ and PLATEL (*Journ. des Mal. Cut. et Syph.*, December, 1899) assert that affection of the fingers in this disease is not so rare as is generally supposed. It usually affects the metacarpal or middle phalanges of the fingers, and is very rare on the thumb. The authors believe it to be due, in every instance, to a direct infection of the finger, traumatism being only of secondary importance in its etiology. It differs from herpes elsewhere in the absence in the beginning of local congestion, a large, flat bleb being the first lesion that is noted.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Cirrhosis and Multiple Adenomata of the Liver.—SCHMIEDER (*Virchow's Archiv*, 1900, vol. clix., p. 290) reports a case of multiple adenomata of the liver with accompanying cirrhosis, and discusses the relation between simple hypertrophy, adenomata, and carcinomata. He also expresses his belief as to the factors giving rise to adenomata of the liver.

In his review of the literature he shows that a sharp line between hyperplasia of hepatic tissue and adenomatous formation cannot be drawn, and that many of the reported cases of adenomata of the liver are combined with cirrhosis of that organ.

Schmieder gives a full clinical history of his case and detailed study of the liver, both macroscopically and microscopically. Careful search reveals to him no connection whatever between the interlobular bile-ducts and the adenomatous formations. On the other hand, he proceeds to demonstrate that the islands of liver cells which are cut off and included by the connective tissue in the cirrhotic process are the points of origin of multiple adenomata. In the centre of these isolated masses of hepatic tissue the liver cells take on a vicarious growth, and one can recognize that the most centrally lying cells are largest. Their structure corresponds completely to normal liver cells, with the exception that they are larger. Single and multinuclear giant cells are found here, and it is by the division of these giant cells that adenomatous cells arise.

His sections of very small adenomata reveal frequently the transition of large multinuclear liver cells into columns of adenomatous cells. Once these columns have arisen the subsequent tumor growth is very rapid, and soon all trace as to its origin is completely lost.

He also finds in isolated patches of large adenomata a change in character

of the tumor which approaches cancer. This is shown by thick columns of tumor cells assuming an alveolar arrangement, by the centre of the growth becoming converted into a mass of cells devoid of definite arrangement, and, finally, by the actual demonstration; in a small area, of true cancerous growth.

Schmieder regards the cirrhotic process as preceding the adenomatous formations, and advances in support of his view the fact that cirrhosis is a diffuse condition, occupying all parts of the liver, and is, consequently, found in portions where adenomata do not exist; that his microscopical study convinces him that cirrhosis is an extremely chronic process, while the tumor formation is a recent and rapidly developing one; and, finally, the clinical symptoms pointing to cirrhosis extended in his case over a period of two years.

In conclusion, he emphasizes his conception that cirrhosis is the first factor in the production of adenomata of the liver, and that the establishment of this view would give a means by which the multiple adenomata might be classified in a special group; at the same time he warns against the assumption that the tumor formation in this case may be placed parallel with the origin of hepatic tumors in general.

Structure and Origin of the Cavernomata of the Liver.—SCHMIEDEN (*Virchow's Archiv*, 1900, vol. clxi., p. 373), in a well-written article emanating from Orth's laboratory, discusses at length the five theories so far proposed in regard to the origin of the cavernomata of the liver, and on the basis of the study of thirty-two cases coming under his own observation adds a sixth theory, in favor of which he advances strong arguments based on histological findings and embryological considerations.

Cavernomata of the liver have frequently been the object of investigations ever since they were first described by Virchow. The interest taken in them stands in no relation to their clinical and pathological significance. They are frequently found at autopsies, but have no bearing on the other lesions of the liver or of the body in general. The point in dispute is their histogenesis; the developed tumor possesses little interest.

The five theories of the starting-point of these cavernous formations are the following:

1. Primary growth of connective tissue.
2. Vascular congestion.
3. Primary atrophy of liver cells.
4. Biliary congestion.
5. Hemorrhage.

Of these theories only two deserve serious consideration, namely, the first and second. The other three have little or no evidence in their favor.

The origin from a growth of connective tissue is the oldest theory, and is at present strongly upheld by Ribbert, who uses the liver cavernoma as a model for all cavernous angiomata, and regards it as an angiofibroma. Against this view is the fact that liver angiomata occur with no connective tissue, but with only liver cells between the blood spaces. It may also be asked why the connective tissue takes the well-known form of the liver cavernoma instead of the structure of cirrhosis or of a fibroma, and when the growth of the tumor stops after a certain time?

The theory of congestion, lately defended by Scheffen, is that the blood-vessels in circumscribed areas of liver tissue become dilated, and that the liver cells in such areas disappear. Against this view are the facts that cavernomata are no more common in cases of chronic passive congestion than in normal livers, and that no evidence in the way of pigmentation can be found favoring the view that the liver cells have undergone atrophy and disappeared.

The writer was started in his work by the study of a case which suggested a defect of development as the origin of these tumors. This led him to study the livers of the new-born. The livers were hardened in formol-Müller and then cut into thin sections. In two cases liver cavernomata were found.

The 32 cases studied in all included some cases from animals other than man. They were divided as follows: 18 solitary and 14 multiple angiomata. In 24 cases the septa were fibrous or fibro-muscular; in 8 cases the partition walls consisted of more or less changed liver cells. In two of these eight cases there were many examples of both kinds which macroscopically could not be told from one another. Most of them lay directly under the capsule.

In the majority of the cases the outer wall was of the same strength as the septa within. Vessels of all sorts accompanied by the ordinary periportal connective tissue entered the tumor, but extensive capillary anastomosis with the vessels of the adjoining liver tissue was lacking.

The writer thinks that the liver cavernoma owes its origin to a developmental anomaly or defect (Anlagefehler), that it belongs in a group with the other tissue malformations occurring in the liver and in other parts of the body, and should be included in group 1 of the classification given by Lubarsch—that is, among the tumors in which the arrangement of the tissue elements differs from the tissue from which they arise, but usually show little or no evidence of growth.

The liver cavernomata do not belong in the same class with the cavernous angiomata of other organs; for example, of the skin. While the latter are to be regarded as true bloodvessel tumors, the liver cavernomata can be regarded as tumors only in a broader sense of the word.

The term "angioma hepatis" is not appropriate; "cavernoma" or "nævus cavernosus hepatis" is to be preferred.

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